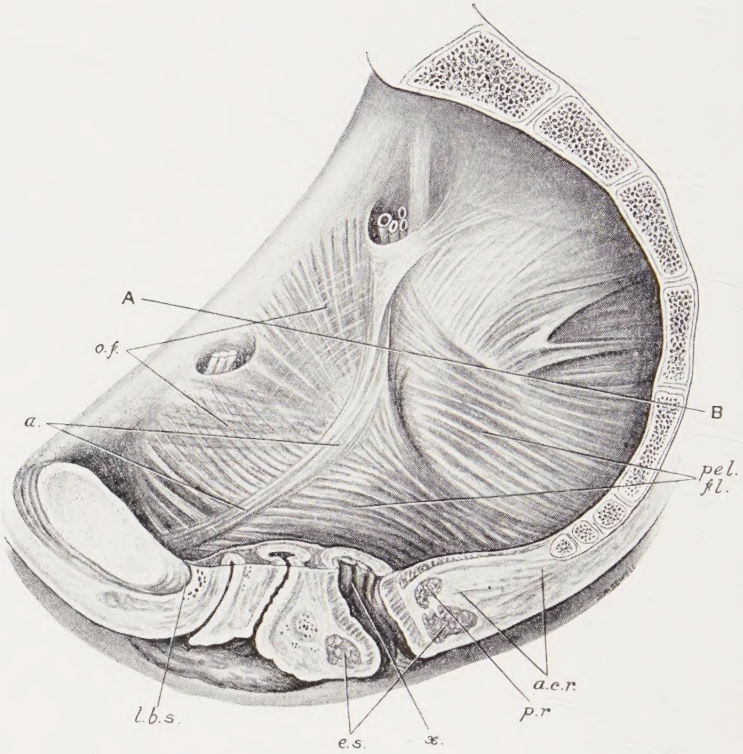
THE STATICS OF THE FEMALE PELVIC VISCERA

"Wherefore I hold that the Matrix is retained in its natural Place and Situation by the Ambient Parts, its Adhesion thereunto,* and its own bulk. Just as the Intestines are in their proper Place by the Environing Parts without the Help of any Ligaments."—JEAN ASTRUC, *Diseases of Women* (Eng. Trans.), p. 272, 1743.

A recent modification of the old view is an apotheosis of the *levatores ani*."—FOTHERGILL, "Prolapse," *Eden and Lockyer's New System of Gyn.*, vol. ii. p. 656, 1917.

* For "its Adhesion thereunto" read "its Pressure therefrom": reference to the original shows this is intended.—R. H. P.



THE PELVIC CAVITY IN THE NORMAL IN THE ANTERO-POSTERIOR PLANE.

Note that the pelvic cavity in this median plane resembles a section of a parabola, the long axis of which (A—B), instead of being vertical, passes much more backwards than downwards. (After Halban and Tandler.)

o.f. = obturator fascia.
a = white line.
pel.fl. = pelvic floor.
l.b.s. = lower border of symphysis.
x = position of perineal flexure. (Clinically this is found to be even nearer to lower border of symphysis than shown.)
e.s. = external sphincter of anus.
p.r. = posterior commissure of pûbo-rectalis muscle seen in section.
a.c.r. = ano-coccygeal raphe.

[*Frontispiece*

THE STATICS OF THE FEMALE PELVIC VISCERA

VOLUME I

IN WHICH THE EVIDENCE OF PATHOLOGY, PHYLOGENY AND ONTOGENY, AND CLINICAL INVESTIGATION, ETC., IS SURVEYED

BY

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TO THE MEMORY OF
MY BROTHER
GORDON
WHO FELL
AT LOOS
SEPTEMBER 25, 1915

PREFACE

THE subject of which this book treats has been before my mind since the summer of 1907. Since that time, opinion has gradually shifted from an absurd and untenable hypothesis to a radical view. This is being more and more held ; and applied practically. But from certain quarters, periodic publications have appeared in which this latter view is dismissed without adequate consideration, and endeavour made to promulgate the other—and in my opinion, erroneous—view.

The present volume treats of the subject from the academic standpoint. I had intended to add chapters on The Evidence of Surgery ; and, perhaps, to say something on The Treatment of Prolapse—from my point of view. Much of this, as a matter of fact, is already written, and indeed is in type. Owing, however, to the amount of time absorbed in the necessary literary research, and more especially to my entry into the Army, I have found it impossible to proceed at a satisfactory rate, and therefore thought it best to present this part of the work, which is self-contained, and to leave to the future the remaining part.

The illustrations have been obtained from various sources, and in each the source has been acknowledged. Most have come from Halban and Tandler's book. A few are original. One I borrowed from *Quain's Anatomy* ; others have come from other works. Figs. 23 and 24 were drawn from a specimen in the R.C.S. Museum, by kind permission of Professor Keith. To all the authors I

express my indebtedness. Most of the drawings have been executed by Mr. Sewell; and I thank him for his skill and pains.

Finally, I have to thank my publishers. They have shown me much consideration; and helped me not a little in this somewhat arduous work.

R. H. PARAMORE.

THE MILITARY HOSPITAL,
GRANTHAM,
March 1918.

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Magna est veritas et prævalebit

THE

STATICS OF THE FEMALE PELVIC VISCERA

CHAPTER I

INTRODUCTION

In a succession of papers, a few years back, I showed that the female pelvic viscera are retained within the pelvis and are thus "supported" by the pelvic floor, which occludes the bony pelvic outlet, forming an obturator for it [68–73]. This pelvic floor is a composite structure, consisting of a bilateral musculature, arranged symmetrically about a median osseo-fibrous raphe—the coccyx and ano-coccygeal raphe or ligament—which extends forwards from the terminal part of the sacrum and ends anteriorly at the posterior limit of the pelvic floor aperture—a deficiency in the pelvic floor anteriorly. The bilateral musculature consists of the levatores ani and the coccygei muscles, which arise from the antero-lateral pelvic walls—from the pubes in front, where the musculature reaches to about half an inch from the mid-line of the symphysis (or less); from the fascia covering the obturator internus muscle, the so-called "white line"; and from the ischial spine. The fibres pass backwards and inwards with varying degrees of obliquity, the anterior ones passing more

backwards, the posterior more inwards; and are partly inserted into the median osseo-fibrous raphe and the terminal part of the sacrum. (See Fig. 8, p. 32.)

The anterior free margin of the levator ani forms the lateral boundary of the pelvic floor aperture—the posterior boundary of this aperture being formed by the junction of this part of the levator ani with the corresponding part of the muscle of the other side. The posterior free margin of the levator ani overlaps the anterior free margin of the coccygeus of its own side; and the posterior free margin of the fan-shaped coccygeus overlaps the small sacro-sciatic ligament. Thus, except for the deficiency anteriorly in the pelvic floor, this floor fits accurately into the diamond-shaped outlet of the pelvis. Above this floor, the pelvic viscera embedded in connective tissue are related to it; below are the superficial tissues with the perineal muscles: and through the aperture in front pass the rectum, vagina and urethra to quit the pelvis and to gain the surface of the body.

The reasonableness of the view upheld becomes obvious when we regard the pelvic viscera with their contents and adventitious parts collectively, and recognise they are continuously being squeezed into the pelvis. For, clearly, under such conditions, a mechanism occluding the wide and diamond-shaped bony outlet is necessary to retain the viscera within the pelvis. And since the pressure with which they are squeezed downwards is very largely due to the thoracic diaphragm and abdominal wall musculatures—the contraction of which even during rest is continuous and during activity is very greatly increased, and which by compressing the abdominal viscera causes a pressure in the abdomen, freely transmitted through the brim into the pelvis—it is not surprising the mechanism occluding the pelvic outlet which opposes this descent should be composed of the same sort of tissue as that which causes

it. For the pelvic floor musculature, like that of the abdominal wall and thoracic diaphragm, is connected with and controlled by the nervous system; and thus the very conditions exist whereby the mechanism occluding the pelvis below, and that compressing the abdominal viscera above, can be combined in harmonious and simultaneous activity, giving rise by their concerted action to the maintenance of a pressure equilibrium, and can further all react together for other purposive ends.

The deficiency or the aperture in the pelvic floor, situated medianly and anteriorly, abutting upon the pubic arch, through which the visceral canals pass to reach the perineum, is obviously a necessity. Even though the pelvic floor (as defined) were admitted by all to occlude the pelvic outlet, and to prevent visceral descent, it would still have to have this deficiency within it. That it should have this deficiency and still act as a pressure-bearing superficies, is not more strange than that the thoracic diaphragm, in spite of its similar action, should be deficient where the œsophagus pierces it. Clearly, this latter does not prevent the diaphragm from playing its important part in causing and maintaining a difference of pressure in the thorax above it and the epigastrium beneath; nor does the deficiency in the pelvic floor in the normal detract from its efficiency as a pressure-bearing mechanism. Surely, it will be admitted that Nature is cunning enough to produce such a combination. Indeed, the passage of visceral canals from the pelvis to the surface of the body through this musculature, affords another and perhaps the determining reason why the pelvic outlet is occluded by a musculature. For since these canals have of necessity at times to be considerably dilated, according to the volume of the visceral contents to be passed; and since, moreover, their place of exit is on this account a weak spot in the parietes, at which visceral parts could easily be extruded, a mechanism simul-

taneously capable of being adapted to the volume of these canals, by being stretched or not so stretched, and of safeguarding this region both during their functioning and at other times, is also obviously essential. Striated muscle is the only tissue which could afford such protection under such conditions; and thus it is no surprise to find the part of the occluding musculature related to these visceral canals at their exit from the pelvis is structurally adapted to this end. An adaptation of the musculature in this region has occurred whereby it forms with the pubic arch a sphincteric mechanism, comparable in every way with the sphincteric arrangement of those muscle fibres of the thoracic diaphragm which embrace the œsophagus; and it acts in a similar way, preventing the extrusion of pelvic viscera (prolapse), just as this other prevents the extrusion of visceral parts in the epigastrium (not merely the regurgitation of stomach contents) into the chest. (See Fig. 6, p. 24.)

Such a conception of the pelvic floor has arisen slowly, and is by no means generally recognised as the true state of affairs. It depends on a recognition of the intra-abdominal pressure and its variations: on the fact that the viscera within the abdomen are compressed by the abdominal wall and thoracic diaphragm. This compression occurs not occasionally, but constantly; but the compression produced is not continuously the same. During resting states, it undergoes rhythmical variations more or less regular: during activity marked excursions occur, often irregular and of great intensity [74]. The recognition of these pressure conditions and variations has also come tardily, and even to-day seems to be realised only by a few. But their importance is obvious. For whilst the knowledge that the abdominal viscera are being constantly pressed downwards towards the pelvis

explains for us the closure of the pelvic outlet by the levator ani muscles, in the absence of this knowledge such an idea could not obtain, and some other explanation for these muscles had to be found. In early days, the levator ani muscles were thought to be elevators of the rectum or anus, supporting the gut as Sappey says [86], and being especially important during defæcation; but they were not thought to support all the pelvic viscera, collectively. Indeed, the pelvic viscera were not thought of collectively at all. Each organ was thought to have its proper place and form, irrespective of the existence of other organs and of the physical conditions to which they are exposed; and thus, factors of paramount importance in the maintenance of the position of any one viscus were overlooked.

Before the time of Schultze [90], the position of the uterus was considered something distinctive, to depend upon the uterus. Its long axis was supposed to be more or less vertical or inclined somewhat forwards in the pelvic axis; and the bladder to be always spherical, however little urine it contained. Schultze was the first to show that when the uterus is in its normal position of anteversion, its anterior surface is always in contact with the bladder; and that though the bladder is more or less spherical when distended, as it empties it becomes flattened from above downwards, the uterus changing in position, remaining in contact with the superior bladder wall, as it does so. Previously, it had been thought these surfaces were not in contact; and in many diagrams in old books the uterus is drawn with its body separated from surrounding parts by a considerable interval. The cervix was supposed to be more or less fixed in the "floor of the pelvis," and to form the base from which the uterine body rose in the cavity and whereby it was maintained in its erect position.

From such a conception perhaps the misleading idea is descended which still prevails—that the cervix, vagina

and bladder form part of the pelvic floor. Indeed, by the term " pelvic floor " some authors include all the soft parts found in the region of the pelvic outlet : not only visceral parts above the pelvic floor (as I have defined it), but also subcutaneous tissues and even the skin, and especially the perineal body have been included. But even those who regard the cervix as part of the pelvic floor have had to hunt round for a means whereby it is supported in its place. It is true the pelvic floor musculature itself depends on the bony pelvis for its position, but the pelvis and it form the walls and floor of a container ; the cervix and its connections are part of the contents. As we shall see, the cervix uteri with its attachments are quite incapable of resisting the visceral downthrust ; it is obvious the bladder could not do so. Clearly, the nervous regulated musculature, placed beneath the visceral parts, is much more suitable ; for it presents a broad superficies to them collectively. In virtue of its structure, by varying in its contraction in response to nervous stimuli, and so becoming harder or softer as occasion requires, it not only can react proportionately to the force of descent, but what is equally important, it can react sufficiently rapidly to meet the great and often sudden changes in pressure which throughout life are continuously recurring. The connective tissue attachments of the pelvic viscera, as of the cervix, are obviously quite incapable of such response ; nor indeed are they fitted by their structure to play such an important part.

But in the absence of the knowledge of such pressure variations, these objections do not obtain. The uterus, possessing an independent position in the pelvis, not pressed upon on all sides by surrounding viscera, nor all these pressed down collectively by visceral parts above, was thought to be maintained in this position by structures which suffice to overcome the effect of gravity merely—its weight only

had to be overcome. The broad and round ligaments were first thought to effect this. Next, the vagina, conceived as a hollow cylindrical tube with more or less rigid walls, and to be more or less vertical, was thought to sustain the cervix in position, thus supporting the uterus like a bust on a pedestal*; and even when it was recognised that the anterior and posterior vaginal walls are in apposition, it has been thought to act as a rigid stem preventing the approach of the cervix to the vulva.† ‡ In 1833, Boivin and Duges [10] gave reasons for rejecting this supposed rôle of the vagina: they were the first to think the utero-sacral ligaments (described first by Baudelocque, 1781) [8] suspend the cervix—a conception which Schultze also firmly believed in. In recent years, the favourite view has been that the "ligaments" in the bases of the broad ligaments are the effective agents—those thick and strong bands (so they are described) which pass from the cervix towards (but not to) the pelvic walls [59, 100], with which are associated the thickenings about the uterine blood-vessels§ and which have to be cut through for the operation of hysterectomy. And some of the authors who have advanced this opinion, have imagined the uterus is placed vertically above the vagina, and that if it were not for these fibrous

* "Hence, as the body of the uterus, in a natural state, rests upon the upper extremity of the vagina, into which its mouth and cervix are projected for a considerable way, that, and not the ligaments, must be considered as the principal instrument for supporting it in its proper place." Manning, 1771 [60].

† "The vagina is one of these supports: the uterus rests on it as on a firm though elastic pedestal, and receives from it no little support indeed." Milne, 1871 [63].

‡ Van der Hoeven, 1909, states that the tone of the vaginal walls plays a part in the maintenance of the uterus in its normal position and in preventing prolapse [42].

§ Fothergill lays stress on the fibrous sheaths of the blood-vessels and accompanying structures which supply the pelvic viscera. He says: "The one constant essential factor in the causation of prolapse is relaxation of the perivascular sheaths" [27].

connections of its cervix, the uterus would descend into the vagina and invaginate its upper part [14]. (Fig. 1 ; see also Fig. 13, p. 61.)

This latter view has been ardently supported and its claims urged upon gynæcologists. These thickenings, it is stated, not only maintain the uterus in position, but their relaxation is the cause of prolapse ; operative treatment, consequently, should be directed towards their repair. As a corollary to this view, the pelvic floor musculature is either neglected or thought to be a degenerate structure. Its thinness is thought to militate against its functioning ; it can have no effect as a means of supporting the viscera—all it can do is to assist the superficial sphincteric muscles of the perineum in the occlusion of the rectum and vagina.* It is urged that the pelvic floor muscles are but the vestigial remains of tail-moving muscles of the simian ancestors of man, and that with the degeneration and loss of the tail, the tail-moving muscles have also degenerated (Fothergill [27]).

That the posterior parts of the pelvic floor musculature in man, though thin, are not functionless, is shown by their remarkable hypertrophy in pregnancy [75] : and their thinness in the non-pregnant is adequately explained by the structure of the abdomino-pelvic cavity. For the pelvis is but a small recess of the large abdomen, and consequent on physical laws, the resistance of the visceral downthrust on to the pelvic floor, which the contraction of the extensive abdominal wall and thoracic diaphragm causes, can be successfully withstood by a muscular diaphragm opposing this downthrust, in spite of the fact that it opposes it with a force considerably less than the force of the compressing musculatures. We have in the abdomino-pelvic cavity a mechanism comparable to that

* Some writers have even thought the superficial muscles of the perineum support the levator ani !—e.g. Van der Hoeven [42].

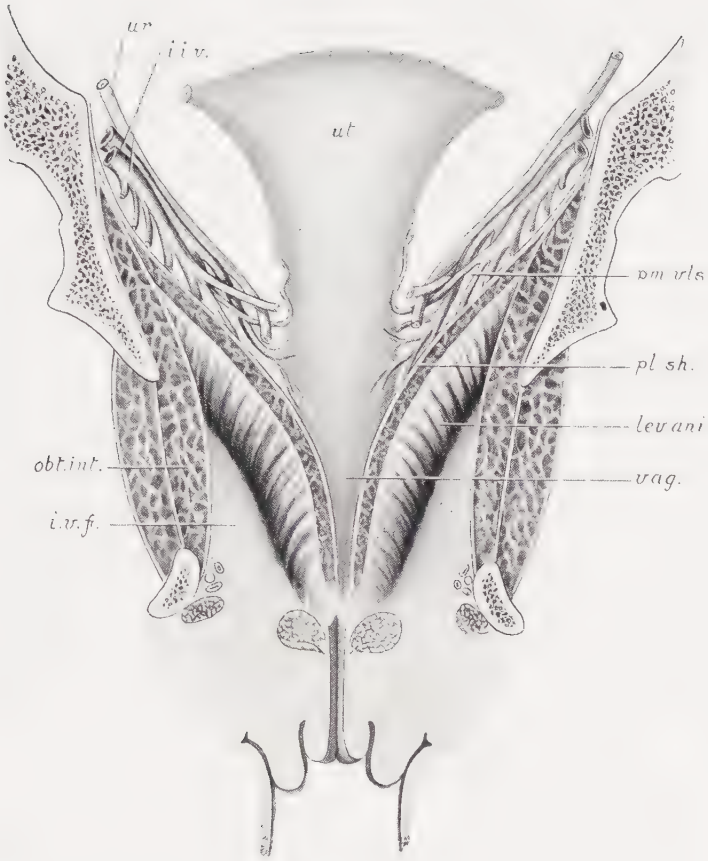

FIG. I.—A VIEW OF THE PELVIC FLOOR AND OF THE UTERUS AND VAGINA AND OF THE MEANS BY WHICH THESE VISCERA ARE SUPPORTED ACCORDING TO CAMERON [14] AND FOTHERGILL [28] FROM IN FRONT.

The uterus and vagina are represented as standing vertically in the pelvis, the parametric blood-vessels passing to the cervix and adjacent part of the vagina being the supposed means by which this position is maintained.

The levator ani muscles, lined internally by parietal pelvic fascia—the pelvic sheath—are shown as forming a steep-sided funnel. Though represented as being markedly muscular, the idea conveyed is unfavourable to the view that these muscles support the viscera in the pelvis.

Note that the lower limit of these muscles in the drawing is on a level with the descending rami of the pubes leading to the ischial tuberosities: and that this is in line with the erroneous idea that the anus is below the level of the ischial tuberosities (see footnote, p. 19). The direction of the muscular fibres, from before backwards, is more or less correctly shown. In a somewhat similar figure (conveying the same idea) appearing in Eden and Lockyer's book on Gynæcology (1916, p. 566) the fibres of the levator ani muscles (again markedly muscular) are represented as passing obliquely downwards and inwards from the lateral pelvic walls to the vagina, in the coronal plane—i.e. at right angles to their real direction. A similar figure appears in anatomical works—e.g. by Poirier and Charpy (82), and apparently originated from Luschka.

(After Cameron.)

ur. = ureter.
i.i.v. = internal iliac vessels.
i.v.f. = ischio-rectal fossa.
pm. vls. = parametric vessels.
pl.sh. = pelvic sheath of levator ani.

To face page 8]

of the force-pump. The small outlet is the pelvis, the piston is the pelvic floor musculature, the medium (instead of water) consists of the incompressible* abdomino-pelvic contents, the work to be done is the successful resistance of the pressure produced by the combined contractions of the abdominal wall and thoracic diaphragm, so that an adequate pressure within may be maintained. The only difference, apart from the medium, between these two mechanisms is that whereas the piston in the force-pump moves and thrusts water into the larger cavity and thus raises weights, in the body the pelvic floor musculature, except during such visceral acts as defæcation and coughing, etc., remains more or less stationary, preventing descent of the pelvic viscera, and by so doing maintains the pressure within at the level for the time being in question; and its activity—that is, its resistance—is increased or diminished according to the pressure that is brought to bear on it by means of the medium which is compressed by the contraction of the other body-wall musculatures. The work done in the body is a compression of this medium—the abdomino-pelvic contents—causing the intra-abdomino-pelvic pressure [74]. Thus, it follows that the anterior abdominal wall musculature and that of the diaphragm must be stronger and better developed than the pelvic floor musculature; for they are much more extensive—they form nearly all the walls of the much larger part of the abdomino-pelvic cavity. So the fact of the relative thinness and weakness and apparent inefficiency of the pelvic floor musculature is not a reason for rejecting it as inefficient and functionless; but it is just what physical laws would lead us to expect.

And in my lectures on the Evolution of the Pelvic Floor [72], I showed how erroneous is the argument on which the second objection is based. For some animals with long

* Except for the gas in the intestinal tract.

and actively mobile tails, such as the horse and the cow, do not possess muscles homologous with the levator ani of other mammals; whilst in other animals in which the tail is absent, such as the guinea-pig, the so-called abductor of the tail is conspicuously present. And in the higher apes and in man, though the tail has been lost, the levator ani itself persists. I showed that these pelvic floor muscles, in all animals which possess them in varying extent, exist to sustain a visceral pressure produced within the abdomen by the abdominal wall and diaphragm adequate for the needs of the animal in question: I showed that there is reason to believe the visceral pressure is different in different animals according to their manner of living; and that the end desired is achieved by the pelvic-floor muscles drawing the root of the tail towards the perineum, serving the same purpose as they serve in man, the only difference being one of degree. The tail-moving muscles are the intrinsic muscles of the tail, namely, the sacro-coccygei and their continuations. These, it is true, in man have degenerated.

Whilst the thickenings in the bases of the broad ligaments have rightly called for comment, and their origin and significance need inquiry, it is to be pointed out that their existence and their function (if any) can never be explained, either by studying them in the dead, or at any one moment of time in the living. The life processes with which they are concerned and which possess duration must be considered and reviewed; the part they play *during periods of time* in the living must be the basis of the research, for they also undergo a process of "becoming": the consideration of the mere fact that they are difficult to cut through at operation can teach nothing except that they are difficult to cut through—it does not and cannot show their significance.

And it is strange the position of the uterus should be considered as though it were an isolated organ; and stranger

still that the interest attaching to the causes of its position should have been so keen as to exclude a consideration of the forces at work in the maintenance of the other viscera in their places. For one would think that what supports the one supports the other—that the same kind of forces are in evidence in each case. And this is all the more remarkable since the connective tissue in which the bladder and rectum are embedded possesses no such thickenings passing from these viscera towards the pelvic walls. Such are as inconspicuous and difficult to find as the ligaments of the cervix—so it is stated—are easy to demonstrate. It may be admitted that these uterine ligaments have all the appearances of being capable of supporting the *weight* of the uterus, that indeed they could support it, were the uterus an isolated organ—just like a spider's web can support the spider [34]. But in reality they are incapable of such action, just as the spider's web can bear no more than the spider's weight ; for the uterus is not an isolated organ, but there is a considerable pressure bearing upon it. And this is the essential objection to this view. These so-called uterine ligaments—and the same applies to the perivascular connective tissue—are unable to withstand the frequent and considerable strains which occur within the pelvis, and which the ligaments would have to oppose unassisted if they served the purpose imputed to them.

There is, I am glad to say, evidence that some gynæcologists have considered these reasons to be valid, and have accepted the principle that the pelvic floor musculature plays at least an important part in the visceral support. But many are still sceptics. Some see in certain conditions arguments against the muscle ; and in other conditions arguments for the ligaments. Some of these we are to consider, in the hope of finding a solution to this problem.

CHAPTER II

THE UTERUS A FLOATING ORGAN

It has been advanced by some gynæcologists that the uterus cannot be supported by the pelvic floor, because the uterus does not touch the floor, does not rest upon it; but that in the normal, the lowest part of the cervix is situated half an inch or so above the floor.* † Besides, the uterus moves in the pelvis just as though it were suspended and had no relation to the floor, like a swing from the ceiling or a hammock between lateral supports. Similarly, it is supposed that the easy mobility of the ovaries militates against this conception. If the ovaries are supported by the pelvic floor or by other parts of the pelvic walls, according to the position of the individual, why do they slip about so easily?

It seems to have been assumed by those who make these objections that the female genital organs, if they are supported by the pelvic floor, should be related to it like a vase on the mantelpiece—in which case there is no difficulty in recognising the factors producing equilibrium. The fact that the physical conditions about the uterus in the pelvis, and its consequent relation to the pelvic floor, are quite different from those of such an example, is over-

* Eduard Martin says: "der Uterus bei der stehenden Frau nicht der unpaaren Levatorplatte aufruht. . ." [61]

† And Hastings Tweedy says: "The upper edge of the levator muscle is situate an inch below the fixation point of the cervix —a situation which precludes the cervical position from being dependent on the muscle" [100].

looked. That the physical conditions are different is easily perceived. In the body, the uterus is surrounded by displaceable organs approximating to its own specific gravity, and therefore itself is easily displaceable: the vase on the mantelpiece is surrounded by air, also displaceable, but of much less specific gravity than the vase, which is for this reason much less easily displaceable.

If a uterus is removed from the body and placed on a table, its relation to the table becomes similar to that of a vase on the mantelpiece. Plainly, to be supported by the table, the uterus must touch, must rest upon the table. Moreover, the physical conditions become such that the uterus is much less mobile than in the body; it is relatively immobile, like the vase on the mantelpiece. And the reason of this is, the uterus is now surrounded by air—by a medium, that is, of much less density than the tissues amidst which it is naturally surrounded.

If we take a uterus and place it in a vessel (such as a specimen jar) containing fluid (e.g. water), the uterus immersed in the fluid will sink to the bottom of the vessel and rest on the floor; and the effect of its mass on the floor, where it touches the floor, will correspond with the difference in the specific gravities of the uterus and fluid. It will be much less than in air. It will indeed be so slight that the gentlest touch from below will cause it to bob upwards or slip from side to side, just in the same way as a child's toy balloon is so readily displaceable. And the reason of this easy mobility is the same in each case—the mobile body is possessed of a specific gravity approximating to that of the medium in which it is moved, and is not obstructed by surrounding bodies nor by ties to fixed structures which plainly must limit the movements—that is, the medium consists of particles easily displaceable. Similarly, in the latter months of pregnancy, the gentlest touch suffices to displace the fœtal head within the lower

uterine segment, in spite of the fœtal mass approximating seven pounds or more in weight (when measured in air), because the fœtus is immersed in a mobile fluid approximating to its own specific gravity. The effect of the immersion is to annul the fœtal weight in its relation to the liquor amnii, because gravity attracts the liquor amnii itself downwards with approximately the same force as it pulls on the fœtus; and since the particles of the medium are easily displaceable, the slightest extraneous force on the fœtus is sufficient to upset the existing equilibrium. Yet the weight of the fœtus, as of the amniotic fluid and placenta, has to be borne, and of course is borne by the walls and floor of the abdomino-pelvic cavity.*

In the body, the pelvis and abdomen act as container, and the uterus and other pelvic and abdominal viscera completely fill it, and since the latter have approximately the same specific gravity as the uterus, since they are to a certain extent displaceable, it is obvious the physical relation of the uterus to the pelvic floor upon which the viscera press, will be similar—in so far as the visceral parts about the uterus are displaceable—to the physical relation of bodies immersed in fluid media of about their own specific gravity to the floors of the vessels containing them, such as we have cited—to that of the uterus immersed in liquid to the floor of the jar containing both, to that of a toy balloon immersed in air to the ground or other support on which it rests, to that of the fœtus in the liquor amnii to the lower uterine segment. Slight extraneous forces will upset the existing equilibrium, and readily cause changes in the position of the uterus. Forces will effect this which, were the uterus lying on a table immersed in

* Because the body under natural conditions is immersed in air. When immersed in water, as when bathing, the weight of the viscera is taken off from the body walls, and is borne by the pressure of the water. The body walls are thus relieved of part of their work.

air, would be quite inadequate to produce any change in its position ; because in this case the specific gravities of the uterus and of the medium are so greatly different, and consequently gravity has a correspondingly increased effect or power of "gluing" the body of relatively high specific gravity to the support on which it lies. Thus, the pelvis being full of viscera, and the parts about the uterus being easily displaceable, the conditions in the body are such as to give to the uterus an easy mobility and to simulate suspension of the organ from above.

CHAPTER III

THE SHAPE OF THE PELVIS AND THE CLOSURE OF THE PELVIC FLOOR APERTURE

Such a figure will immediately raise objections. On the one hand, it may be said that the pelvis and pelvic floor do not form such a container; on the other, that the visceral parts about the uterus, even collectively, do not comprise such a medium. And it may be objected that, even if they do, since the specific gravity of the uterus is perhaps a little greater than that of the parts about it, it ought to have sunk so low in the pelvis as to have come as close as possible to the pelvic floor—the cervix ought to have sunk down the vagina, as it does in the early stages of prolapse. It is suggested the reason the uterus does not touch the pelvic floor is because it is suspended by the ligaments we have mentioned. It is imagined that the pelvic walls and the pelvic floor form the sides of a funnel with a large hole at the lowest part; and that the sides of the funnel possess considerable steepness. It is known the cervix is some little distance from this aperture, sometimes so far back as to be difficult to reach, as for example in fat patients; and it is supposed if the ligaments did not suspend the cervix in its normal position, it would slip down the funnel and out through the aperture (see Fig. 1, p. 8). Besides, there is this difference between the uterus in the body and the experimental cases of mobility cited—that whereas the uterus, when displaced from its normal position in the

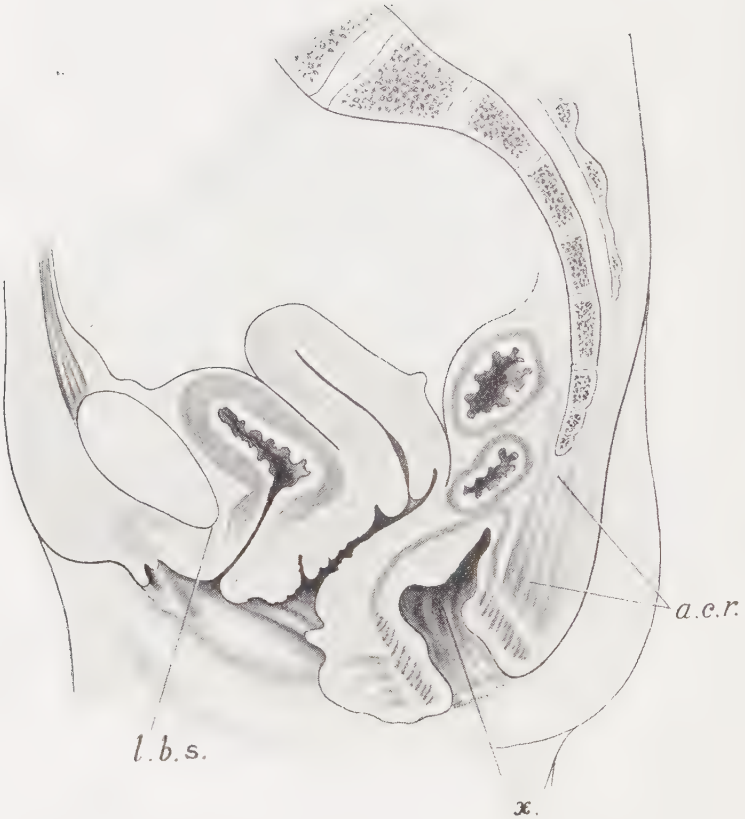

FIG. 2.—SECTION OF THE PELVIS, SHOWING DESCENT OF THE PELVIC FLOOR AND THE SIMULATION OF A FUNNEL OWING TO POST-MORTEM RELAXATION OF ITS MUSCULATURE.

Note the obliquity of the post-anal part of the pelvic floor, which is almost vertical; and the relation of the perineal flexure of the rectum to the pubic arch. In the normal, these two parts are in close proximity. (From Quain's *Anatomy*.)

a.c.r. = ano-coccygeal raphe. l.b.s. = lower border of symphysis.
x = perineal flexure of rectum.

[*To face page* 17

body, returns to that position when the displacing force, e.g. the vaginal finger, is removed; displacement of the uterus poised in water is not followed by a return to its original position, but some other resting position is assumed. It is true the fœtal head, displaced in the liquor amnii by a finger in the vagina (ballottement), does return to its former position; but this presumably is due to the limited room in the pelvis for the dilated lower uterine segment, and the condition of the non-pregnant uterus in the pelvis is different. Although here external forces (e.g. gravity) may perhaps play some part in the return of the uterus to its normal position after displacement, surely, it may be argued, this return is to be explained by the ligamentous connections of the cervix.

These objections must be considered; we shall endeavour to show they are imaginary or untenable, and that such a parallel as we have drawn can be maintained; and we will deal first with the conception that the pelvis with its floor forms a steep-sided funnel inadequate to function as such a container. This conception is erroneous. It is based on sections of the dead (Fig. 2). In these, owing to the post-mortem relaxation and elongation of the muscle fibres, a considerable descent of the pelvic floor with increased obliquity of the coccyx and enlargement of the pelvic floor aperture has resulted; and thus a funnel is simulated. Similar conditions are found in the living, in cases of ptosis of the pelvic viscera (without prolapse, and to be observed in both sexes), due to an atrophic condition of the pelvic floor musculature; but are best marked when this is complicated with extrusion of the viscera—that is, when the aperture is more considerably dilated. In these cases, especially when the disease has been of long duration, structural changes in the bony pelvis itself have generally also resulted: the concavity

of the sacrum, for instance, may be found much reduced. Since the coccyx forms the skeleton of the pelvic floor, to which a part of the pelvic floor musculature is directly attached, it is easily intelligible that instead of occupying its usual position, it should have become more or less vertical according to the yielding of the musculature inserted into it, and to the position assumed by this, the post-anal part of the pelvic floor. Such pelves, especially with their soft parts in place, are typically funnel-shaped (Fig. 3).

But in the healthy living female (and male) quite different conditions obtain (see Frontispiece). In these, the outline of the pelvic cavity in the mesial sagittal plane, so far from presenting the appearance of a funnel, is curvilinear, and approaches a semicircle. It is not a semicircle, however; for it is distorted by being bulged disproportionately in one direction. But this bulging—strange to say—is not antero-inferiorly, in the region of the pelvic floor aperture; but posteriorly, in the region of the sacrum. Whilst the sacrum is highly concave; the pelvic floor, instead of dipping downwards in a funnel-like way, is straightened out, displaying but a gentle curve as it passes from the sacrum forwards to the pelvic floor aperture and by the sides of the aperture to the pubes. The musculature, because tonically contracted, is taut; and in the normal remains so. Thus, instead of a funnel leading to the aperture, the pelvic cavity in this median plane resembles a section of a truncated cone (or parabola), the long axis of which (passing through the brim approximately through its centre but obliquely to its plane), instead of vertically downwards, passes much more backwards than downwards, and abuts upon the fourth or the junction of the fourth and fifth sacral vertebræ, as represented by *A*—*B* in the Frontispiece. This line, produced in the opposite direction, passes through the hypo-

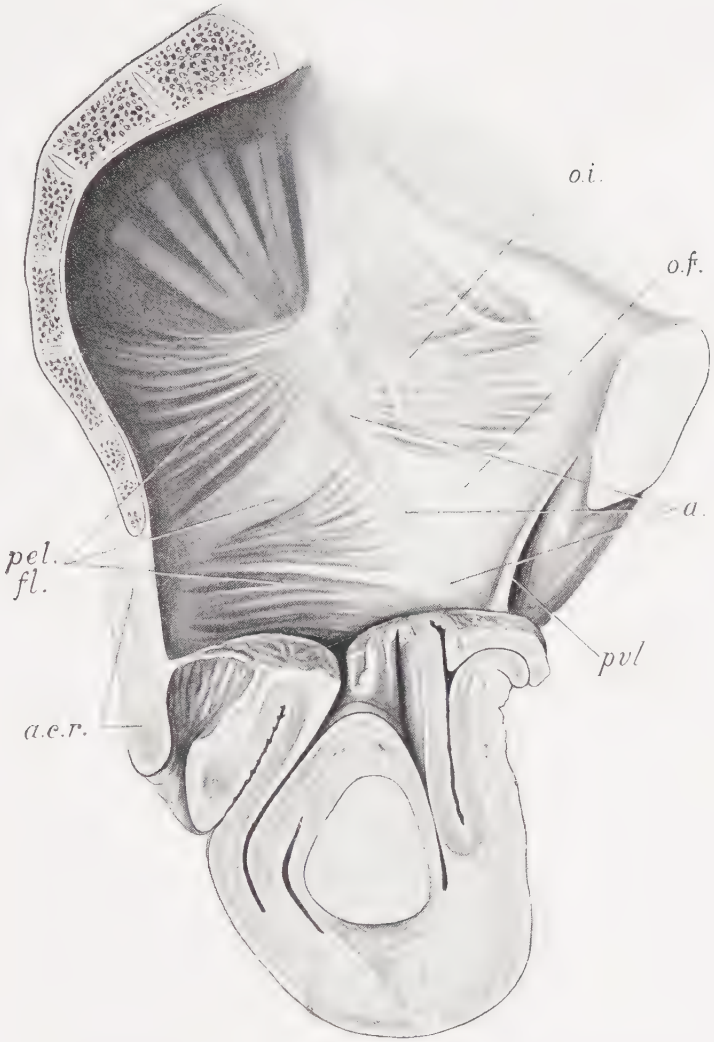

FIG. 3.—SECTION OF A PELVIS FROM A CASE OF ADVANCED PROLAPSE, SHOWING THE TRANSFORMATION OF THE PELVIC CAVITY INTO A FUNNEL FROM PATHOLOGICAL CAUSES.

(After Halban and Tandler.)

The coccyx and the ano-coccygeal raphe (a.c.r.) are almost vertical. The pelvic floor musculature (pel. fl.) is atrophic: much of its muscular tissue has been replaced by fibrous tissue, which is seen between the individual muscles, and to a lesser extent between the fibres of each muscle. The inner layer of the obturator fascia (o.f.), by which the pelvic floor is attached to the pelvic brim, is thin, allowing the obturator internus muscle (o.i.) to be seen through it; and it is much elongated, especially in front, so that the white line (a), instead of being attached anteriorly to the pubic bone has sunken much beneath this part. Posteriorly, the attachment of the white line to the ischial spine has scarcely altered.

Note the elongation and thickening of the pubo-vesical ligament (p.v.l.).

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gastrium and strikes the lower part of the abdominal wall, and is a relation not without significance.

That the pelvic floor in the median plane occupies the position stated is seen at once by inspecting the position of the anal groove, which is an index to the position of the median antero-posterior raphe of the floor. When a normal individual is examined in the lateral position, the summit of the anal groove is out of sight, the two buttocks are in apposition, and completely hide it: to display the summit of this groove, one has to separate the buttocks. The skin and subcutaneous tissues covering the median raphe of the floor are obviously highly placed; they are only gently curved, being besides straightened out not far removed from the horizontal. They pass only gently downwards to the anus, which is situated above the level of the ischial tuberosities.* But when the pelvic floor is lower than normal, when it has been caused to descend by the causes which produce marked prolapse, the anal groove presents as a furrow and is seen without any separation of the buttocks; and its summit, instead of a line, possesses some breadth. It is also much more obliquely inclined, passing much more downwards, often displaying considerable steepness; and the anus is on a much lower level. Similar evidence is obtained from tracings of the external superficies of the pelvis in the median antero-posterior plane. These can easily be made by means of a leaden wire, adjusted in the anal groove to its summit and continued forwards to the pubes and backwards to the sacrum. Such tracings of the normal are curvilinear. They do not altogether represent the outline of the pelvic cavity, they are much more nearly semicircular because of

* Unhappily, in representations of the normal, it is not at all uncommon to see the anus depicted far below these tuberosities. See, for example, the drawing on page 203 of the *Journal of Anatomy and Physiology*, Vol. 42, and compare it with the drawing on page 99 of the same volume.

the increasing thickness of the sacrum superiorly; but they do indicate the pelvic cavity is not like a funnel held vertically. And palpation of the floor between two digits—i.e. with a finger within the vagina or rectum and a thumb on the skin externally in the anal groove—will show that there is not much disparity between the shape externally and internally in this plane and region. What disparity exists is in the region of the aperture. Lateral exploration by the finger within the vagina similarly will show that the lateral halves of the pelvic floor musculature do not form steep sides of a funnel as stated. Posteriorly, at least between the ischial spines, there is no indication of a funnel. Inspection of coronal sections at right angles to the brim confirm this impression (Fig. 4). Although the sides of the pelvic cavity are steep; at their junction with the floor, at least posteriorly, the outline inclines rapidly inwards, forming a shallow and stable concavity. It is only in front, where owing to the encroachment of the obturator internus muscles upon the pelvic space, to the narrowing of the bony pelvic outlet which in this region occurs, and to the existence here of the pelvic floor aperture, the collective effect of which is to determine a diminution in breadth of the levator ani muscles in this part, that a funnel is simulated.

Examination of this aperture by means of transverse sections through the pelvis at right angles to the plane of the aperture show that on either side of the aperture the pelvic floor musculature forms a shelf facing upwards and inwards on which the lateral parts of the bladder rest; and that it has all the appearances of a truncated funnel or inverted cone. But here, besides having little lateral breadth, the musculature is modified to form a sphincter for the aperture, and the appearance of a funnel—that is of a body with oblique walls enclosing a lumen—is misleading. The figure from Halban and Tandler (Fig. 5, p. 22) shows

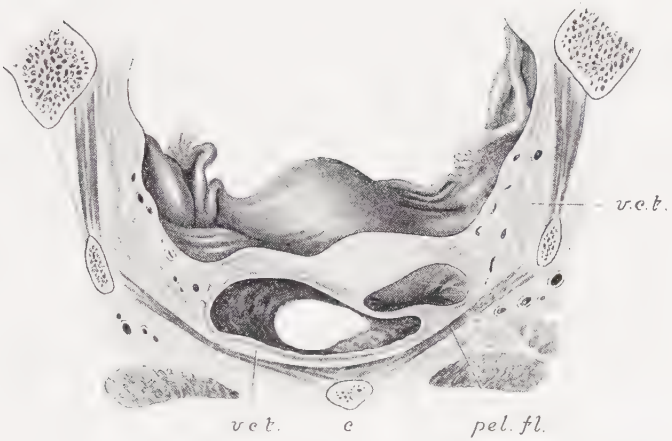

FIG. 4.—TRANSVERSE SECTION OF THE PELVIS THROUGH THE ISCHIAL SPINES IN THE NORMAL, SHOWING THE PELVIC CAVITY IS TRULY BASIN-LIKE IN FORM.

(After Halban and Tandler.)

c. = coccyx; pel. fl. = pelvic floor; v.c.t. = visceral connective tissue.

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what a thick bundle of muscle fibres the levator ani forms in this region: behind the aperture, between the ischial spines, the muscle is represented as a relatively thin sheet, but in the region of the aperture a peculiar development has taken place. Palpation of the muscle in the living confirms this difference: the muscle about the aperture is much more conspicuous than that arising from the ischial spines.

Dissection shows that the levator ani here really consists of two distinct parts. The one (the pubo-coccygeus) is more or less in the same plane or superficies as, and continuous posteriorly with, the rest of the pelvic floor musculature (ilio-coccygeus and coccygeus); the other (the pubo-rectalis) is placed just below this in quite a different plane. Both arise from the pubic bones more or less horizontally, their most median fibres reaching internally along the bone to a point half an inch from the mid-line of the symphysis, so that the width of the aperture—i.e. the distance from the inner edge of the muscle bands on one side, to that of those on the other—is one inch.

The upper band—the pubo-coccygeus—passes backwards and inwards and slightly upwards; and is inserted into the ano-coccygeal raphe or into the coccyx (some say, the sacrum) [97]. With its fellow of the opposite side, it forms a V-shaped muscle; the apex of the V being attached to the coccyx, the separated arms arising from the pubes, the interval between forming with the pubes the upper part of the pelvic floor aperture through which the rectum, vagina and urethra escape from the pelvis (Fig. 8).* But the lower band—the pubo-rectalis—which also passes backwards by the sides of the vagina and rectum, instead of being attached to the skeleton of the tail (coccyx) or to the ano-coccygeal ligament, turns inwards behind the ano-rectal junction of the gut, and becomes continuous

* Facing p. 32.

with the corresponding muscle layer of the other side, the two muscle arms together forming a U-shaped muscle. The posterior commissure of the muscle, formed by the bend of the U, consists of striated muscle: there is no tendon or fibrous connection intermediate between the muscle arm of one side and that of the other; but the muscle fibres of the one run into those of the other (Fig. 9, p. 33). And this commissure of the pubo-rectalis —it is to be emphasised—unlike the apex of the V-shaped pubo-coccygei, has no connection posteriorly with the ano-coccygeal raphe or with the coccyx or sacrum: it lies free in loose fatty connective tissue, immediately below the level of the main part of the pelvic floor musculature, but above the level of the superficial perineal muscles, and so above the external sphincter of the anus, part of which is inserted into the coccyx. Its muscle arms, extending forwards to just above the inferior border of the pubic arch, form with that part of this resisting structure between their origins the lower and much more important part of the pelvic floor aperture; for through this also the three visceral canals, collected together in close proximity, have to pass before they can gain the surface of the body. This they only reach after traversing the subcutaneous tissues, in which they diverge, and in which their terminations are surrounded by sphincter muscles and come into relationship with other superficial muscles.

It is clear from the difference of their anatomical relations that the effect of the contraction of the pubo-coccygei and of the pubo-rectalis must be different. In the case of the pubo-coccygei, their contraction must pull the coccyx more forwards, flexing it, if possible; and since the muscle arms become more taut they must compress bilaterally to some extent the visceral canals placed between. Cripps [15] has shown that the rectum is constricted in this way; and Hildebrandt [39] thought the aperture in the pelvic floor is

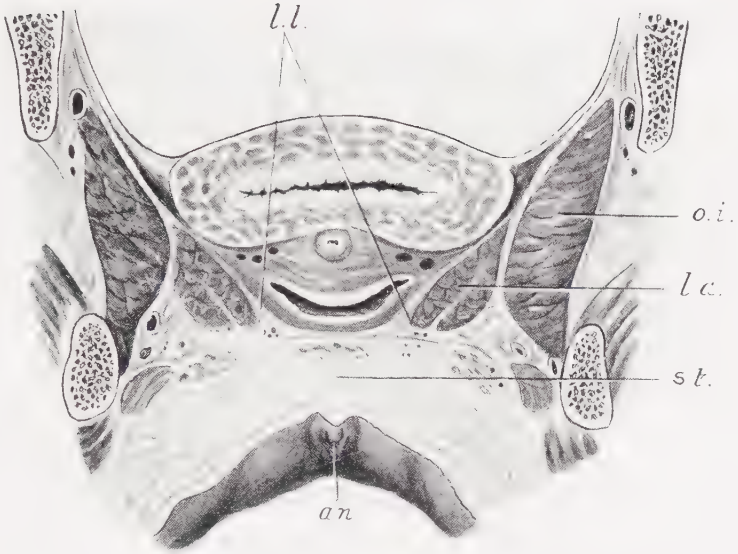

FIG. 5.—TRANSVERSE SECTION OF THE PELVIC CAVITY THROUGH THE PELVIC FLOOR APERTURE IN THE NORMAL, IN WHICH A FUNNEL IS SIMULATED, AND THE MECHANISM OF THE CLOSURE OF THIS APERTURE SCARCELY APPEARS.

(After Halban and Tandler.)

Compare this figure with Figure 1, facing p. 8.

l.l. = lateral limits of pelvic floor aperture.
o.i. = obturator internus muscle.
l.a. = levator ani. (Note that the muscle fibres are cut transversely: they pass from before backwards almost horizontally.)
s.t. = subcutaneous tissue which here forms what is known as the perineal body.
an. = anus.

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occluded by this means—the only mechanism obtaining in many lower animals (Fig. 7, p. 29). But in the case of the pubo-rectalis, owing to its freedom behind from attachment to the skeleton, the result of its contraction is different. It can have no effect on the coccyx, but for this very reason it acts much more efficiently as a sphincteric mechanism; for it is able to pull the rectum at its flexure, and with it the ante-posited vagina, forwards and to compress them against the pubic arch, not only occluding them, but also the aperture in the pelvic floor through which they pass (Fig. 6). Thus we find that, whilst the larger part of the pelvic floor musculature is so arranged that its visceral superficies is placed upwards in apposition with the collective mass of pelvic viscera; the muscle arms of the pubo-rectalis, although horizontal anteriorly where they arise from the bone, gradually change in this respect, and become vertical posteriorly where they join each other behind the rectum. The visceral superficies of its posterior commissure is placed more or less at right angles to the rest of the pelvic floor; and presents its broad surface to the gut as this passes downwards and backwards to the skin. Thus, just as the main mass of the pelvic floor musculature is admirably adapted to oppose the visceral downthrust, by its superficies being placed more or less at right angles to this downthrust; so it is obvious the best possible conditions are afforded by the adaptation of the pubo-rectalis for the occlusion of the aperture in the pelvic floor by a compression from behind forwards of the visceral canals.

There is plenty of evidence to show that the action of the pubo-rectalis compresses the visceral canals in this way. We instinctively and vigorously contract the muscle to prevent untimely defæcation. It not only compresses the gut antero-posteriorly, but increases the normal perineal kink. By its action, solid fæces can be retained even when

the external sphincter has been torn through, and is thus rendered useless, as in complete rupture of the perineal body. Because of this action on the rectum, Holl called the pubo-rectalis the sphincter of the rectum [43]. Its action on the vagina is similar; for the vagina at this level is placed between the rectum and pubes, and when the muscle compresses the one it compresses the other. Thus the muscle plays an important part during coitus (Hildebrandt [40]), sometimes causing retention of the penis within the vagina —penis captivus—as Hildebrandt, Budin [13] and others have given instances; and according to Dickinson [20], the muscle is especially well developed in erotic women and in patients suffering from painful lesions about the vulva and anus, e.g. fissures (causing vaginismus), as well as in muscular women, that is, in those accustomed to hard work, as young domestics, and in women with wide pelves.

That the pubic arch plays a prominent part in this mechanism is shown by the marked indentations which were produced anteriorly in the wax cylinders Budin used to investigate this closure of the vagina. Impressed by Hildebrandt's paper, Budin investigated the sphincteric action of the levator ani by inserting a cylinder of wax, 37 mm. in diameter, into the vagina, the muscle being voluntarily relaxed, and then getting the patient to contract or draw up the parts as much as possible. After subsequent relaxation the cylinder was removed. Examination showed an impression had been formed in the wax, which, whilst completely encircling the cylinder, was most marked anteriorly, corresponding to the lower border of the pubic arch. Measurement of the constricted part showed that the antero-posterior diameter, instead of 37 mm., measured only 26 mm., and the transverse diameter 35 mm. In another patient, who was pregnant, the contraction was more powerful still; the antero-posterior diameter of the resulting constriction being found to measure 24 mm.,

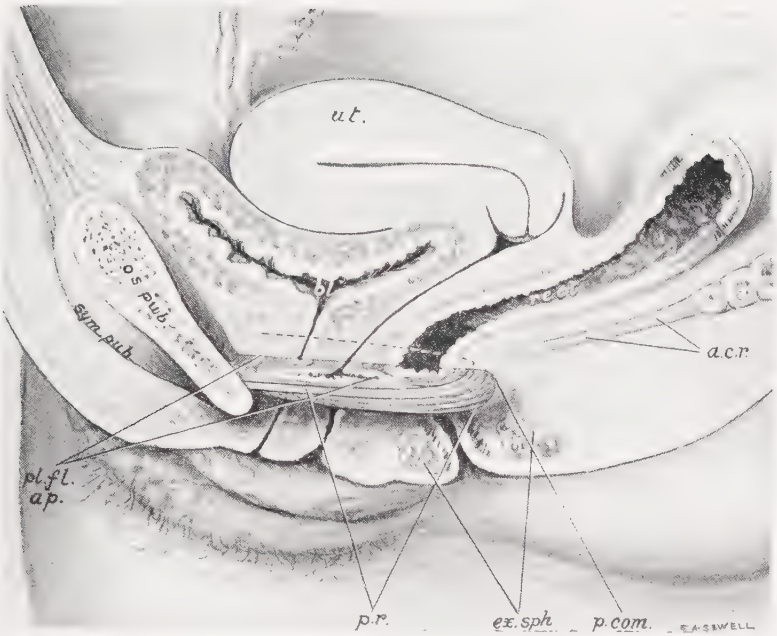

FIG. 6.—A LATERAL VIEW OF THE VISCERAL CANALS PASSING THROUGH THE PELVIC FLOOR APERTURE IN THE NORMAL, SHOWING THE MECHANISM OF THE CLOSURE OF THIS APERTURE BY THE PUBO-RECTALIS.

The urethra, vagina and rectum, in a median sagittal section of the pelvis, are seen passing through the pelvic floor aperture (pl.fl.ap.) formed by the pubo-rectalis which lies in a horizontal section. One half of the muscle (p.r.) is seen this side of the section; whilst the other half, passing forwards behind the section, is indicated by the interrupted line. The pubic bone (os. pub.) is cut through on this side of the median plane of the pelvis, and from its lower border the pubo-rectalis of this side arises. Note that the muscle passes backwards by the sides of the urethra, vagina and rectum, and turns in behind the gut to become continuous with the other half of the muscle the other side.

sym. pub. = symphysis pubis.
ex. sph. = external sphincter of anal canal.
p. com. = posterior commissure of the pubo-rectalis.
a.c.r. = ano-coccygeal raphe.

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and the transverse diameter 32 mm. instead of 37 mm. as before.

This experimental work of Budin, which has been confirmed by Dickinson, shows that on active contraction of the levator ani muscles, the aperture in the pelvic floor, previously distended, may be considerably reduced, and that the reduction takes place for the most part in the antero-posterior diameter of the aperture. This is caused by an advance of the posterior commissure of the pubo-rectalis towards the pubic arch, which determines the constriction or compression and occlusion of the rectum and vagina already referred to. The reason why the impression, posteriorly and laterally, was less deep than in front, is to be found in the fact that whilst the muscle fibres form a fairly broad band at the sides and behind, the anterior limit of the aperture formed by the inferior margin of the pubic arch is sharp (Budin). Budin adds that it is certain a much greater constriction than that found in the cases reported would have been obtained by using a substance less resistant to the muscular contraction than the fairly firm modelling wax, and which at the same time had possessed the quality of preserving its resulting form.

It has already been mentioned that the transverse diameter of the aperture, at least anteriorly, at the origin of the muscle bands from the bone, is about an inch (i.e. 25 mm.). When the aperture is not dilated, its widest part cannot be much greater than this. Its antero-posterior diameter, as measured from the mid-point of the symphysis to the posterior wall of the gut at the ano-rectal juncture, is in the resting position (left lateral) about an inch and a half.* Probably it is a little less than this, for the method employed in the measurement may have dilated it a little: and besides, the figure given is the estimate of the probable length from measurements of

* See Appendix to Paper on "The Pelvic Floor Aperture" [73].

several cases, and in some of these it was less, viz. an inch and an eighth. When we think of these measurements, we get the idea of the aperture as a patent foramen in the pelvic floor through which visceral parts could escape fairly easily. But we must remember that the urethra, vagina and rectum leave the body cavity by means of this aperture; and as they do so, since they collectively form a mass of tissue, they more or less block the aperture in virtue of their bulk, but whether they do so or not depends also on the muscle. For even when the passages are empty, since the tissues of which their walls are formed possess volume, being composed of incompressible (though deformable) cells, it is clear they can form an obturator for the aperture. This obturator is kept in place by reason of its circumferential adhesion to the muscle boundary of the aperture—that is to say, the lateral walls of the vagina and rectum are adherent to the pubo-rectalis muscle *; and further the urethra, vagina and rectum are all adherent to each other. By means of this obturator and the sphincteric mechanism about it, the aperture in the pelvic floor is occluded.

The size of the aperture obviously must vary with the size of the obturator. When this is increased by the extrusion of bodies within the canals to the outside of the body –e.g. by the passage of the child during labour and of fæces during the lesser labour of defæcation—the aperture is of course correspondingly dilated and the stretching of its muscle offers corresponding difficulties to the passage. But in the ordinary resting condition, as also during activity, that is when the passages are empty, the obturator is sufficiently embraced to prevent visceral extrusion, the danger of which depends on the pressure within the pelvis, that is, the pressure to which the viscera

* "The non-striped muscle of the rectum and vagina interweaves with the striated fibres of the pubo-rectalis."—Henle [36].

are subjected. When this is increased, the compression of the obturator becomes greater, because the muscle contracts more strongly; when the intra-abdominal pressure becomes less, the compression of the obturator is diminished, because the muscle about it contracts less strongly.

At first sight, perhaps, this is a little difficult to realise. It is so easy to pass a finger into the vagina. So also, is it easy to pass an œsophageal tube into the stomach!—one feels no constriction as the tube passes the diaphragm, and yet a sphincter about the œsophagus is there. The finger or the tube acts as a wedge, and the force each exerts laterally is much greater than the force necessary to cause either to advance. But the force they exert laterally is not a great force—for the dilator is small; it is, however, sufficient to overcome the striated muscle about the one or other canal, with a consequent and corresponding enlargement. But in well-developed nulliparæ and especially in well-developed primægravidæ—in women, that is, in whom the hymen has been torn, but the pelvic floor aperture has not been permanently widened by the injuries consequent on childbirth—one often finds some difficulty in passing two fingers into the passage simultaneously.* One finger has to be passed and the second insinuated alongside the first. The finger already within the vagina presses the posterior vaginal wall backwards to dilate this part, which of course is accompanied by a corresponding dilatation of the pelvic floor aperture —that is, by a stretching of the muscle embracing the visceral canals. In multiparæ, this is usually so easy and offers such little resistance as to be scarcely perceived. But in many primægravidæ, one is sensible that the fingers within the vagina are embraced by something about the vagina constricting them; and especially when the

* Thus, Aarons suggests the use of one finger only in the examination of nulliparous women [1].

patient turns on her back for bimanual examination, the vaginal fingers may be felt to be pressed upwards (in this position) against the sharp lower border of the pubic arch which is readily perceived. And we have to remember that the pressure in the pelvis when these examinations are made, especially in the lateral position of the patient, is low —becoming greater, however, in the supine position : and that under these conditions there is no necessity for great contraction of the muscle, so that the canals it embraces can be correspondingly easily dilated to such an extent.

The aperture, after all, in the resting condition, is not very large ($1'' \times 1\frac{1}{2}''$). It is probably smaller than the measurements given. Such as it is, its length was measured under conditions similar to those in which vaginal examinations are made, i.e. with a low internal pressure. Whether it measures less during activity and violent movements, I cannot say : probably it measures less.* Certainly the canals suffer a greater compression : their cells, however, are deformable, and tissue fluids and blood may be squeezed out from amongst them, which admits of an increased constriction at the level of the aperture. Anyway, the experiments of Budin and of Dickinson, and the clinical experience of Hildebrandt and others, show that the compression exerted by the muscle may be very considerable. And we know that when the muscle is contracted maximally, as in vaginismus, it is extremely difficult or impossible to pass even one finger into the vagina. Finally, we find this sphincter muscle greatly hypertrophied in pregnancy,† just as we find the

* The aperture which in the resting condition is more or less oval in shape, the sagittal diameter being the longer, probably becomes more circular when the pubo-rectalis is maximally contracted : the sagittal diameter diminishing, the transverse increasing—the canals being empty.

† Compare Budin's measurements on a pregnant woman already mentioned in the text.

pressure in the rectum (indicative of the intra-abdominal pressure) permanently raised during the major part of this condition [75].

If we reflect on the biological significance of this

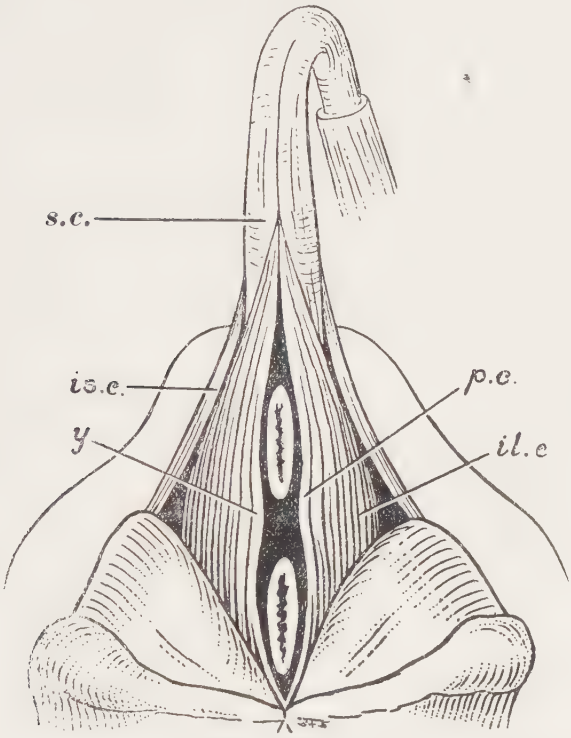

FIG. 7.—AN EXTERNAL VIEW OF THE PELVIC FLOOR OF THE DOG, SHOWING THE VERTICAL MESIAL SLIT, FORMED BY THE POSTERIOR FREE MARGINS OF THE LEVATOR ANI MUSCLES (PUBO-COCCYGEI).

Note that the slit extends from the mid-line of the Symphysis Pubis to the mid-line of the root of the tail, and that the Visceral Canals are bilaterally compressed by the muscular boundaries.

p.c. = pubo-coccygeus. il.c. = ilio-coccygeus. s.c. = sacro-coccygeus.

is.c. = ischio-coccygeus. This muscle is attached to the lateral aspect of the tail root, and is separated here from the ilio-coccygeus by the ventral sacro-coccygeus (s.c.).

"y" indicates the position where the pubo-coccygei in the dog are tied together by some fibro-muscular tissue passing from side to side between the vagina and rectum. In the specimen I dissected, this, though distinct, was poorly developed, and not comparable in this respect with the pubo-coccygei muscles. The sinuous median margins of these muscles in the diagram is an exaggeration. When the tail is erect, as indicated, they are taut.

sphincteric mechanism about the pelvic floor aperture, we gain confirmation of this opinion. The pubo-rectalis does not exist in pronograde mammals, such as the dog: but in them the rectum is flattened from side to side by the two pubo-coccygei, the free median borders of which stretch between the mid-line of the symphysis pubis and the mid-line of the root of the tail, and the gut in them runs a straight course—it is truly the straight gut (Fig. 7). But in the course of the further evolution of the pelvic floor musculature, and coincidently with the assumption of the erect posture, the pubo-rectalis appeared—probably segmented off from the lower parts of the pubo-coccygei. And just as the rest of the pelvic floor musculature—the pubo-coccygei and ilio-coccygei—acting on the coccyx and lower part of the sacrum, has dragged these parts forwards; so the pubo-rectalis has dragged the rectum, with the vagina in front of it, forwards toward the pubic arch, causing those anatomical peculiarities we are all so familiar with—the perineal flexure of the gut and the lower curve in the sigmoid course of the vagina. These become readily intelligible when we recognise this action of the pubo-rectalis on the one hand; and remember that the anus is anchored posteriorly to the tip of the coccyx by part of its external sphincter, and that the vagina is also anchored by the connection of its superficial sphincteric muscle with this anal sphincter in the perineal body, on the other.

Now, although the occlusion of the rectum and vagina in man which the activity of the pubo-rectalis causes (because it compresses them against the bone) is clearly a more efficient one than the occlusion of these canals by the bilateral pressure caused by the two pubo-coccygei in the dog—the muscles becoming taut by extension of the tail—it is obvious that such occlusion is not more necessary in man than in the dog: for both the gut and

the vagina, both in man and in the dog, are provided with sphincter muscles—that around the anal canal in both being especially efficient. But the change in the pelvic floor becomes intelligible when we recognise that the pressure in the pelvis has of necessity to be higher in man than in the dog, because of man's erect posture. This dragging forwards of the canals is correlated with the necessity for more efficiently withstanding the down-thrust of the pelvic viscera which has become more intense in man: it is not a means for preventing the escape of visceral contents—this is provided for by the intrinsic sphincters of the visceral canals. Thus, the occlusion of the vagina and rectum by the pubo-rectalis is not an end in itself. Its purpose is to prevent visceral extrusion, because only in this way can the pressure in the pelvis be maintained. For this end, the muscle compresses the visceral canals against the pubes, not to occlude them, but to occlude the aperture in the pelvic floor through which they pass, and which it effects by reason of the bulk of these canals (on cross section) and by the incompressibility of their cells. Whilst it does this, it also subserves other ends; but these are secondary and in man non-essentials (e.g. as in coitus).*

Thus, transverse sections through the pelvic floor aperture at right angles to its plane, when studied alone, give one an erroneous idea of the aperture. It is like making

* It is a remarkable fact that whilst those who maintain the uterus is supported by the thickenings of its connective tissue attachments, and that the pelvic floor plays no essential part in this, yet admit the sphincteric function of this part of the pelvic floor (pubo-rectalis) (see Fothergill, *Manual of Diseases of Women*, 1910, p. 91). This author rejects the idea that the rest of the pelvic floor musculature functions, because he thinks it is a degenerated tail muscle. But it is obvious that if one part of the pelvic floor musculature can play a part in the visceral retention in the pelvis, the possibility that the rest of the musculature may play a similar part must be admitted.

a transverse longitudinal section through the body of the non-pregnant uterus—a tracing of such a section would give the notion that the uterus possesses a large cavity; but sections at right angles to this show it is only a capillary cleft. If we examine a sagittal section through the pelvic floor aperture in the median plane, we see that the anterior and posterior walls of the vagina are in apposition, and the same applies to the rectum when empty. And examination of horizontal sections through the pelvis in the plane of the aperture (i.e. at right angles to the long axis of the body) reveals the visceral canals in relation to the pubic arch and muscles forming the boundaries of the aperture. We see the H-shaped lumen of the vagina, described by Henle [36], caused on the one hand, by the adhesion of the lateral vaginal walls to the muscular boundary on either side of the aperture, and on the other, by the presence of the urethra in front and the rectum behind which bulge the anterior and posterior vaginal walls backwards and forwards respectively, all the parts being in close juxtaposition (Fig. 8). Pirogoff's plates of such sections [81] show the relations well (Fig. 9, p. 33). The parts are all crumpled together, even after death, by the sphincteric mechanism through which they pass. There is no doubt these parts are all squeezed together even when their canals are empty. Why should this constriction occur, if not to occlude the aperture through which they pass?

Since the pubo-rectalis becomes twisted in its course, from in front where it is horizontal to behind where it is vertical, it is obvious it must assume an intermediate oblique position, which in transverse vertical section gives the appearance of the sides of a funnel. It is clear, when the pelvic floor is lowered and the aperture dilated, either by a post-mortem relaxation of the muscle or by a stretching due to weakness in the living, that the pelvic floor

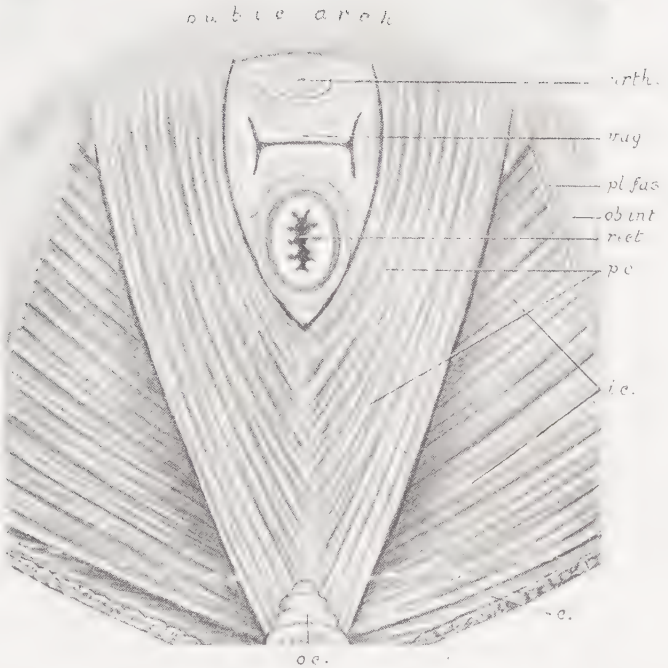

FIG. 8.—A VIEW OF THE PELVIC FLOOR IN MAN FROM ABOVE IN WHICH THE VISCERAL CANALS APPEAR IN TRANSVERSE HORIZONTAL SECTION, A LITTLE ABOVE THE PELVIC FLOOR APERTURE, TO SHOW THE H-SHAPED LUMEN OF THE VAGINA AT THIS LEVEL, AND THE RELATIONS OF THESE CANALS TO THE PUBO-COCCYGEI AND TO EACH OTHER.

(After Henle—much modified and added to.)

pl.fas. = white line of parietal pelvic fascia.
p.c. = pubo-coccygeus } = levator ani.
i.c. = ilio-coccygeus }
ob.int. = obturator internus.
c. = coccygeus.
o.c. = coccyx.

To face page 32]

becomes more or less like a funnel. It may be allowed, perhaps, that the aperture itself is a funnel. But if so, it is a funnel most curiously shaped—very shallow in front in the region of the pubes, but of some little depth posteriorly—and a funnel, moreover, blocked by its normal

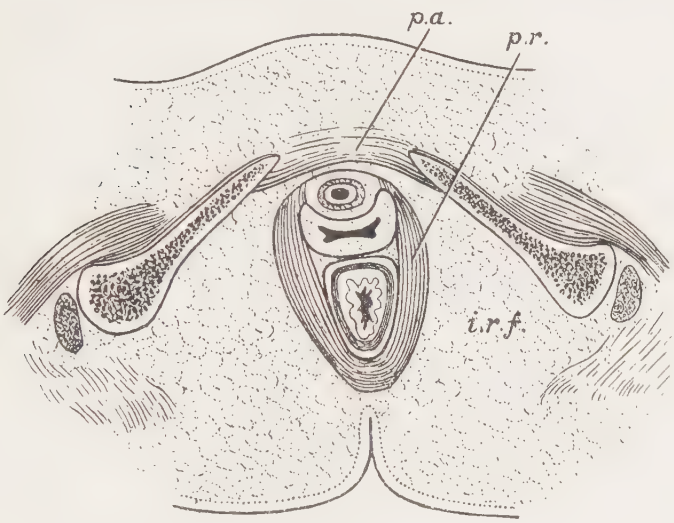

FIG. 9.—A TRANSVERSE HORIZONTAL SECTION THROUGH THE PELVIC FLOOR APERTURE, TO SHOW THE COMPRESSION OF THE VISCERAL CANALS AND THEIR CLOSE RELATION TO THE PUBIC ARCH DUE TO THE ACTION OF THE PUBO-RECTALIS.

The fibres of origin do not appear because the section has passed below their level, but the section has cut through the main mass of the muscle, which is lower than normal owing to Post-Mortem Descent. (From Pirogoff's *Atlas* [81].)

p.a. = pubic arch. p.r. = pubo-rectalis muscle.
i.r.f. = ischio-rectal fossa.

constituents. But to say that the shape of the entire pelvic cavity is that of a funnel, because it has an aperture in its floor, is unreasonable; it is either an abuse of language, or it is a misrepresentation of fact. The pelvis is not funnel-shaped: but is truly basin-like in form.

CHAPTER IV

THE POSITION OF THE PELVIC FLOOR APERTURE AND THE INTRA-ABDOMINAL PRESSURE

1. The Position of the Aperture and the Effect of Gravity: the Superficial Parts

But in the erect posture, the pelvic floor aperture is situated at the lowest part of the pelvic cavity. For in this position, the pelvic floor is obliquely placed. It slopes downwards and forwards in a gentle curve towards the pubes, becoming here almost horizontal. Similarly, in the same position, the upper surface of the pubic bones and symphysis slope backwards and gently downwards to meet the pelvic floor. And here, medianly, abutting upon the pubes, is the aperture in the pelvic floor: and this is its lowest part. Thus, when the individual is standing erect, the aperture is in the most dependent part of the pelvic cavity, and is apparently most dangerously placed. It is certainly most dangerously placed as regards the effect of gravity acting upon viscera just above: and gravity has been considered of very great importance in the statics of the pelvic viscera, having been thought the essential force to be overcome by those structures on which the maintenance of the normal position of the viscera depends.

The reason of this is that in prolapse the visceral parts "come down" through the vaginal orifice when the patient gets about, that is when she is in the erect posture, and that usually, and always in the early stages of the

disease, they return within the pelvis when she goes to bed or lies down. Since gravity invariably attracts bodies downwards, and since the effect of gravity must be different in the two cases, it is supposed that gravity plays the essential part in causing prolapse, that the organs fall down through the vagina in virtue of their weight. Thus, Matthews Duncan, who thought the uterus was normally kept in place by what he called the "Retentive Power of the Abdomen," regarded prolapse as the result of gravity acting on a patient in whom the retentive power of the abdomen had become deficient or obliterated [23]. And of late years, the uterus has been thought to fall through the vaginal orifice because of a relaxation of its connective tissue attachments; especially when it is larger and so heavier than normal—the result of subinvolution, "chronic metritis," or the presence of a small fibroid. But that prolapse is caused by the action of gravity is an illusion, and it is certain an increased weight of the uterus has nothing to do with it. The hypothesis of the retentive power of the abdomen has long since been abandoned: whilst the imputed relation of the elongation of the connective tissue attachments to its inception is a transmutation of cause and effect—it is like putting the cart before the horse. For the uterus does not come down because its connective tissue attachments relax or elongate; but the elongation of the attachments is caused by the descent, by the extrusion of the viscera, that is, by the prolapse.

Matthews Duncan's conception [22] was a very curious one. He supposed the uterus in the normal is suspended in mid-pelvis, like Mahommed's coffin in mid-air, the position of the uterus being caused by the aspiratory effect of the thorax. This view he put forward nearly half a century ago (1868); and, subsequently, he strenuously upheld it. His reputation was so great, that it is no exaggeration to say his teaching enormously influenced

gynæcology in this question, and that it effectually befogged and obstructed the recognition of the actual physical conditions in the pelvis and abdomen. Yet he came to his opinion by a logical process: but his premises were unsound, his examinations imperfect, his observations incomplete. Examination showed him the uterus is a floating organ and is poised in the pelvis. How is this effected?

Matthews Duncan rejected the idea that the round or the broad ligaments cause a suspension of the uterus, because they stretch so easily. And he was unaware of the thickenings in the bases of the broad ligaments, especially the thickenings about the uterine arteries—or he attached no importance to them: had he known of them, he might have rejected them too, for the same very good reason. These thickenings appear to have been referred to first by Savage in 1872 [87], and were described by Mackenrodt in 1895 [59], who called them the ligamenta transversales coli; and, since, considerable attention has been paid to them [77]. The belief in recent years that these basal thickenings suspend the uterus is but the result of finding that to remove the uterus they have to be cut through, and that in the non-pregnant adult they are thick and tough: it is not a result of a study of their biological significance or physiological capacity for such a purpose. And hysterectomy was not often performed in Matthews Duncan's day. Further, Matthews Duncan had no conception of a mechanism occluding the pelvic outlet, supporting the pelvic viscera collectively—of the pelvic floor musculature. All he knew was that an aperture existed in the median line, in the most dependent part of the body, through which the uterus and other visceral parts often prolapsed: and that a knife could be put in at the anus, and brought out through the vagina, and a great big slit formed in the median line, and yet in spite of this the uterus and indeed

FIG. 10.—A VIEW OF THE BUTTOCKS AND PERINEAL REGION OF A NORMAL FEMALE OVER FORTY YEARS OF AGE, IN WHICH THE NECESSITY FOR AN OBTURATOR FOR THE PELVIC OUTLET SCARCELY APPEARS.

Had the woman been a young and robust nullipara, with much firmer subcutaneous tissues and better marked gluteal folds, the necessity for such an obturator would appear much less.

The upper buttock is displaced upwards by the attendant's hand to display the perineal cleft.

[*To face page* 37

other visceral parts (e.g. the bladder) often maintained their normal positions. He thought this showed that the pelvic viscera are not supported from below! Clearly, if such be the case, and if the uterus is not suspended by ligaments, it must be maintained in position by an aspiration from above (Fig. 10).

Matthews Duncan's conception was, therefore, for him, the only possible one. It presupposed that the pressure within the diaphragmatic domes and epigastrium is always negative: and this he maintained and taught, although he never attempted to prove it. Yet he strongly opposed those who ventured to think otherwise. Especially did he oppose those more enlightened but less renowned people, who believed the intra-abdominal pressure is increased in pregnancy—or at least that the growth of the uterus in pregnancy causes an increase of pressure upon the liver and kidneys—and that this is the probable cause or at least is associated with the albuminuria of pregnancy and with the changes in the liver which occur with that toxæmia. And he condemned them [25] because they could not (at least, did not) prove the premises on which their opinion was based, in spite of the similar neglect he was guilty of in relation to his own hypothesis.

For Matthews Duncan's hypothesis was raised on the flimsiest basis: on the fact that in the knee-chest, and allied positions, air easily enters the bladder or vagina; and on the fact that in the intervals between the pains of childbirth, the head of the child, before its greatest circumference is borne, slips back into the pelvis. Both observations are true: but the inferences drawn from them are invalid. In the knee-chest position, we know the intra-pelvic pressure is negative; but this is not evidence that this pressure is negative in other positions of the body, as in the erect posture.* And whilst the

* See Fig. 15, p. 85, and compare it with Fig. 6, p. 24.

recession of the child's head in the intervals between the pains—that is, when the abdominal wall musculature and thoracic diaphragm are not maximally contracted by the straining, and the pressure caused by the uterine contraction has died away—may possibly, if the patient be in Sim's position, be partly due to the same cause—a small negative pressure in the pelvis—a much more potent factor is the elastic resistance of the soft parts occluding the pelvic outlet, and especially of the increased tone of the stretched pelvic floor musculature. For it is clear that this, having been distended by the conical protrusion of the advancing part of the child's head, can, when the expelling force is reduced by the cessation of the pains and the straining, react on this part of the head and cause it to recede.

Nor is the inference that the pelvic viscera are not supported from below, because they may retain their normal positions when the perineal body is torn or cut through, valid. It is indeed an appalling mistake. For what can cause greater astonishment, to any one familiar with the size of the bony pelvic outlet, a hiatus measuring four inches by five (Fig. 11) and cognisant of the existence and magnitude of the intra-abdominal pressure, whereby the pelvic viscera are continuously being pressed downward toward that outlet, than the conception that the perineal body retains the pelvic viscera within the pelvis and is the only means by which they can be supported from below? Yet, again and again, one sees it stated that because rupture of the perineal body is not followed by prolapse, the pelvic viscera cannot be supported by the pelvic floor. Thus, Dr. Riddle Goffe (of New York), at the British Medical Association of July, 1908, is reported to have said that the uterus was supported by its ligaments, not by the pelvic floor: that weight for weight, no other organ in the body had so many ligaments: that when the

support of the pelvic floor was destroyed, as in complete rupture, the uterus remained in place [31]. And many English and Continental gynæcologists reason in the same way. The complete rupture referred to by Dr. Goffe is presumably a complete rupture of the perineal body.

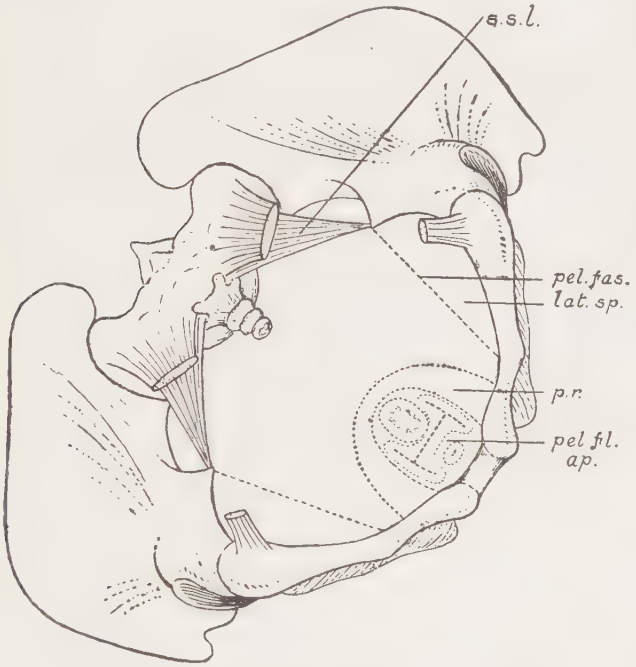

FIG. 11.—A VIEW OF THE OUTLET OF THE BONY PELVIS, IN WHICH THE NECESSITY FOR AN OBTURATOR MEASURING FOUR INCHES BY FIVE BECOMES APPARENT.

s.s.l. = small sacro-sciatic ligament.
pel.fas. = indicates the position of the "white line" of the pelvic fascia.
lat. sp. = space occupied by obturator internus muscle.
p.r. = indicates the position of the pubo-rectalis muscle, which, with the pubic arch, encloses pel.fl.ap., the pelvic floor aperture.

Complete rupture of the pelvic floor would involve a tear from the aperture in the pelvic floor, right back beyond the coccyx to the sacro-coccygeal junction, or

into one or other sacro-sciatic foramen; for that alone would sever all the muscular segments comprising the floor. Such a tear is unknown. Tears in the pelvic floor are rare posteriorly: they usually occur antero-laterally, in the region of the origin of the muscle from the pubes, and involve that part of the muscle bounding the aperture, which is thereby permanently enlarged [89]. Obviously, Dr. Goffe could not have referred to the floor of the pelvis; what he meant was the perineal body—quite a different thing.

Thus, the argument should read: The uterus is supported by its ligaments and not by the perineal body, because the support of the perineal body may be destroyed, as in complete rupture, and yet the uterus remain in place. But the argument is seen at once to be invalid when we think of the large diamond-shaped outlet of the pelvis, and compare the area to be filled in with the mass of the perineal body. The idea is so ridiculous that we do not credit any one with ever entertaining it. Clearly the wide outlet has not been considered: only the median cleft seen clinically is thought of; and of this, the extensive post-anal part is omitted and attention only directed to the anterior part. It is plain that the coccyx and the two inches in extent of soft tissues in front of the coccyx, between it and the anus, into which the ilio-coccygei and pubo-coccygei are inserted, and upon the tone of which the position of this raphe depends, must be considered in this question of the visceral support; and that antero-laterally, at the sides of the aperture, the arms of origin of the pubo-coccygei from the pubes must be similarly considered. As we shall see, we have very considerable evidence for the opinion that these parts of the pelvic floor do support the pelvic viscera collectively; that the pelvic floor is a mechanism retaining the viscera within the pelvis. And in the last chapter we saw reason to

believe that in the region of the aperture itself, where the visceral canals escape, this place of exit is protected by the specially adapted part of the musculature—the pubo-rectalis—the function of which is to prevent visceral extrusion, and the injury to which is to be especially avoided.

The fact that the perineal body may be destroyed without affecting the positions of the pelvic viscera teaches us this—that the perineal body plays no part whatever in the visceral support. Nor can we be surprised, when we come to consider it anatomically or clinically, that this should be so. For it consists of subcutaneous tissue, succulent and loose; in which superficial muscles, derived from the primitive cloacal sphincter, meet; and is covered by skin, its most resistent element. It is that part of the subcutaneous tissue which finds itself between the diverging extra-pelvic parts of the vagina and gut (anal canal). It crept in between these canals when the cloaca divided to form them—a change which caused the evolution from the common cloacal sphincter of sphincter muscles for each orifice. With these parts of the visceral canals, and the superficial muscles, it is placed altogether below the pelvic floor, below the level of the aperture, below the level of the pubo-rectalis guarding that aperture. Obviously, if the pubo-rectalis guards the aperture, and by compressing the visceral canals as they pass through the aperture, prevents visceral extrusion, a destruction of the perineal body below the aperture, provided the pubo-rectalis remains intact, cannot affect the question of visceral support: and, clearly, since the pubo-rectalis passes *behind* the rectum, it need not necessarily be injured when the perineal body is torn. Thus, we learn that when prolapse is associated, as it often is, with rupture of the perineal body, it is not this rupture which has caused or facilitated the prolapse; but an associated

injury of the pubo-rectalis, which obviously is liable to injury by the passage of the child, and which, moreover, clinical examination in such cases of prolapse shows has often occurred.

And examination of the perineal body in well-developed women, in whom it has never been injured, as by child-birth, confirms this opinion. Although displayed as a large triangular mass in anatomical representations of median sagittal sections of the pelvis, palpation with one finger in the anal canal and another in the vagina shows it consists of extremely soft and easily deformable tissues. It is indeed so soft that it can with the greatest ease be flattened into a narrow septum. The only structures to be felt within it are the anterior part of the ring-like external sphincter of the anus, and above this, the corresponding part of the internal sphincter. We can feel no dense bands or structures coursing through it, such as we should suppose we could feel from reading the work of Savage [87], who described the transverse fibrous connections of the perineal body as "real ligamentous bands," which he called the "ischio-perineal ligaments." These, he supposed, passed transversely from one ischial tuberosity to the other, through the perineal body; and thought they acted as a sort of brace, supporting all the tissues above, even the levator ani, the pelvic floor itself! Nor can we feel the so-called tendinous centre, nor the pre-rectal fibres of the levator ani; nor make out laterally the transverse perineal muscles—all of which have been magnified by clinicians into important structures, on the integrity of which the efficiency of the perineal body as a visceral support has been supposed to depend.

It has been thought these transverse perineal muscles act like the fibrous brace of Savage, supporting all the parts above. So also, it has been imagined that the pre-rectal fibres of the levator ani muscles act as a tie between

the levator ani muscle of one side and that of the other *; and that when they are torn through these muscles tend to be bulged apart [33]. Others have thought that the pre-rectal fibres, arising from each side from the pubes and ending in the perineal body, form a sling for the vagina inferiorly, bracing it up towards and against the bone, acting in the same way on the vagina as the pubo-rectalis does on the rectum (Piquand and Hue [80]). Piquand and Hue even attributed to these scanty pre-rectal fibres the constriction of the wax cylinders which Budin used in his experiments, as did Budin himself. If this were so, the pre-rectal fibres should form a muscular mass capable of being perceived on palpation, and much more ought they to be found on dissection. The actual findings disclose that the effective constrictor to which these results were undoubtedly due, is not situated within the tissues of the perineal body; but is the main mass of the pubo-rectalis muscle, which passes behind the rectum, and which forms such a conspicuous fleshy structure that it can be easily and plainly felt. The existence of this, and the fact that as it pulls the rectum at its flexure forwards, it must of necessity act similarly on the vagina, compressing the vagina at this level between the gut and the pubic arch, renders such a special mechanism for the closure of the vagina unnecessary. The fact that all these may be divided by childbirth, by a tear passing from the vagina

* In the dog, there are some definite musculo-fibrous strands, passing from the pubo-coccygeus of one side to that of the other between the vagina and rectum; and these must tend to prevent undue separation of the median borders of these muscles, forming the perineal slit, when, for instance, the dog barks. But the dog has no pubo-rectalis. The pre-rectal fibres of man are, I believe, more evident in the infant than in the adult. They are probably vestigial in origin—the transmitted result of an attempt by nature to prevent visceral extrusion, which gave way to the much more efficient mechanism the development of the pubo-rectalis affords. (See Fig. 7, p. 29.)

into the anal canal, reaching as high as the recto-vaginal septum, involving the perineal body completely, without the subsequent occurrence of prolapse, shows at once that the pre-rectal fibres and the other superficial structures in the perineal body are in the question of the visceral support of no importance and may be entirely neglected.

Nor is the teaching of anatomists in discord with this opinion. All these superficial muscles, we are told (Cameron), except the ring muscle surrounding the anus, are poorly developed, often difficult to demonstrate, and are less easy to find and less definite in the female than in the male. The pre-rectal fibres are scarcely to be found in the adult; such as they are, they resemble fibrous tissue rather than muscle; nor is there any structure passing from the ischium of one side to that of the other through the perineal body that could act as an efficient brace, adequate for the visceral support. The only fibrous structure found is the posterior border of Colles' fascia, which, although better developed in the female than in the male, is yet membranous in nature (Cameron).

Yet even Halban and Tandler state the perineal body participates in the support of the female pelvic organs, and that by the contraction of the pre-rectal fibres the posterior vaginal wall is raised towards the symphysis. Further than this, they believe that the "Urogenital Diaphragm," that thin and ill-defined triangular fibro-muscular layer situated anteriorly within the pubic arch, in a plane immediately below the anterior part of the pelvic floor and its aperture, and which reaches back to the anal canal, is also of importance. They think this diaphragm supports the anterior part of the vagina and the bladder in the region of the pelvic floor aperture; and

attribute the non appearance of prolapse in cases of rupture of the perineal body to this diaphragm being still intact. But when we recognise the existence, relations, mode of action and strength * of the pubo-rectalis, how unnecessary is it to fall back on such a means for the preservation of the pressure conditions in the pelvis and for the visceral retention! When we remember that the arms of origin of the pubo rectalis pass forwards by the sides of the vagina to be attached to the pubic arch antero-laterally to the vagina, and that they are therefore placed at the sides above the lateral parts of the urogenital diaphragm, and that medianly the vagina is occluded by the pressure (of the rectum) from behind against the pubic arch, it is clear that this diaphragm, like the perineal body, lying below this mechanism, does not come in for consideration as a means of supporting the pelvic viscera. Nowhere external to the abdomino-pelvic cavity, within which the viscera are contained by particular muscular sheets evolved for the purpose of containing them, and for containing them under pressure, do subcutaneous structures, whether fibrous or muscular, in spite of passages from the cavity through its walls, play any part in the retention of the viscera.

The urogenital diaphragm is essentially a subcutaneous structure, and comparable in every way with the perineal body. It consists of membranous layers of connective tissue with poorly developed muscles. As the vagina and urethra leave the pelvic floor aperture, they pass through this diaphragm, the most important component of which is the superficial part of the urogenital sphincter (Kalischer [49]). This here surrounds both these canals together in one sweep of its fibres; but only a few of these are

* Farabœuf (quoted from Piquand and Hue [80]) found that a dynamometer placed within the vagina indicated an increase of pressure, which reached 12 to 15 kilogrammes when "these muscles contract." But he attributed this effect to the contraction of the pre-rectal fibres already referred to.

attached to the bone (Kalischer). As its deeper portion is traced upwards, its fibres gradually recede from the vagina and embrace the urethra alone; and this part, according to Kalischer, has no connection with the bone. Even Halban and Tandler say that if this urogenital sphincter be disregarded, the urogenital diaphragm contains but scanty muscle fibres. The fact that the lower part of the anterior vaginal wall can be easily invaginated so that the internal aspect of the pubic bones and symphysis can be completely and satisfactorily palpated, by which examination the tissues of the urogenital diaphragm are displaced and offer no appreciable resistance and by which the muscles within it cannot be palpated, shows at once that these structures cannot be compared with the pubo-rectalis, which is easily palpable antero-laterally and which offers considerable resistance to displacement.

And that the urogenital diaphragm prevents prolapse is also untenable. Its tissues suffer very considerable injury on childbirth, as Halban and Tandler expressly state. For as the fœtal head protrudes from the vulva, all the maternal tissues around, both vaginal and vulvar, are reflected outwards upon its projecting part as a sort of cuff; and whilst the part behind usually suffers most, because it has to bear the strain of deflecting the head anteriorly, the parts at the sides and in front are also greatly stretched and sometimes torn. When the perineum ruptures early, the parts in front doubtless suffer less; but the reason prolapse may not follow in such cases cannot be imputed to a persisting integrity of the urogenital diaphragm, but to the pubo-rectalis having remained intact. Indeed, it is because of such a tear, that the pubo-rectalis often escapes an injury sufficient to impair it. For as the head descends, its advancing part, having dilated the pelvic floor aperture protrudes through the aperture, and comes to bear upon the perineal tissues placed below the

aperture, and to depress and to stretch them. Its effect on these is so great that just before birth, when the head appears at the vulva—at about which time its greatest diameter occupies the pelvic floor aperture, displaced into a plane passing downwards and backwards from the pubes—the distance along the skin from the fourchette to the posterior margin of the pelvic floor aperture (which is beyond the dilated anus) measures no less than 5 inches. The resistance of the perineal tissues deflects the anterior pole of the head anteriorly, and causes the head as it descends to revolve about a transverse horizontal axis, so that the part of the head now within the embrace of the pubo-rectalis from being circumferential becomes elliptical—causing a much greater enlargement of the pelvic floor aperture and a correspondingly increased stretching of the pubo-rectalis. If before this extension of the head has happened, the perineal tissues give way by tearing, the greater stretching of the pubo-rectalis is prevented ; for the laceration allows the head to be born in a much less extended position than otherwise would occur. Thus, instead of the head passing through the pelvic floor aperture obliquely, it passes through less obliquely or even with its long axis at right angles to the plane of the aperture. Thus, the pubo-rectalis, embracing a circumference rather than an ellipse of the head, is less stretched and less liable to be torn. In this way an early perineal tear becomes a blessing in disguise. But if the perineal body is torn *after* delivery of the head, after it has deflected the head anteriorly and caused the head to pass through the pelvic floor aperture obliquely—as may happen and often does happen with the birth or delivery of the shoulders—with the perineal tear may be associated a preceding tear of the pubo-rectalis muscle, and a subsequent prolapse may arise. Thus, to avoid as much as possible injury to the pubo-rectalis in labour, when the

head appears at the vulva, two fingers should be inserted into the vagina in front of the head, between it and the anterior vaginal wall; and as the pains come on, the advancing part of the head should be pulled backwards as much as possible, so as to prevent or at least to lessen its extension. This manœuvre not only saves the pubo-rectalis from a greater stretching; but, to one's infinite surprise, it preserves the perineal tissues themselves, as Toff [99] has stated, from laceration.

Nor does the occurrence of prolapse after symphysiotomy and pubiotomy and after birth in cases of split pelvis show the urogenital diaphragm plays the part imputed to it, as Halban and Tandler contend. For even though the structures in this diaphragm suffer a greater injury than usual as a result of childbirth under such conditions, the pelvic floor aperture is also affected. Birth under these conditions often leaves the pelvis wider than before, the pubic bones with the pubo-rectalis arising from them remain more apart, and even if the pubo-rectalis itself is not lacerated by the delivery, the pelvic floor aperture necessarily is permanently enlarged transversely. This in itself is sufficient to explain the prolapse in these cases.

2. The Position of the Aperture in Relation to Visceral Impactions

Many considerations—anatomical, experimental and clinical—show that a pressure greater than the atmospheric exists within the abdomino-pelvic cavity: that the physical conditions in the pelvis are the very reverse of those Matthews Duncan supposed. The hemispherical shape of the human pelvis is itself an indication of such pressure. For in its phylogeny, the pelvis has approximated to the form of greatest possible capacity with smallest superficies, which distinguishes it from the pelvis of every other animal.

And the change from the fœtal to the adult type, which represents the later phases of this phylogenetic development, is most marked and significant.* The enlargement of the pelvic cavity, especially posteriorly, its change in shape, and the descent of the bladder and uterus, abdominal organs in the fœtus,† into the pelvis, which occurs with growth in the early part of life, is evidence that the pelvic viscera are squeezed into the pelvis and pressed down on to the pelvic floor. Not only does the pelvic floor musculature undergo a great development, but the bony pelvis itself reacts, the sacrum becoming markedly concave. This concavity is the result of two forces: the visceral thrust from above, which is directed downwards and backwards; and the pull forwards of the pelvic floor musculature on the terminal part of the sacrum, and on the coccyx, whereby this from being vertical assumes an oblique position, being directed downwards and forwards.

The existence of the pressure clinically can be shown by measuring the pressure within quiescent viscera. The pressure within the rectum, except in the knee-chest and allied positions, when it is negative, is always considerable, varying with the posture of the subject and being greatly increased by activity in a perfectly intelligible manner. Thus, when standing erect, it measures 20 to 28 mm. Hg.: when lying flat on the back, 10 to 14 mm. Hg.: when on the side with knees flexed, 6 to 8 mm. Hg. On coughing, it rises suddenly to 80 or 100 mm. Hg. or

* In the fœtus, immersed in a medium (the liquor amnii) of about its own specific gravity, a fœtal intra-abdomino-pelvic pressure, caused by the reaction of the muscular walls of its own abdomino-pelvic cavity, is not required as by the adult, immersed in a medium of much less specific gravity (air). For in the fœtus, the pressure of the medium itself will plainly affect it; but in the adult, the pressure of the medium (air) is much less, and consequently, to obtain even the same effect as before, the muscular walls of the abdomen and pelvis have to play a greater, a more intense part.

† See Fig. 16, facing p. 93.

more, and falls as rapidly. On straining, it rises gradually to 50, 60, or 80 mm. Hg. or even higher,* and remains raised as long as the breath continues to be held. During movements against resistances, as during the lifting of heavy weights, its increase is marked. Measurements in the stomach in different postures † show similar variations, but less high values. The fact that the pressure in the stomach (even in the erect posture) has been found by the great majority of observers to be always positive, greater than the atmospheric pressure (except under unusual or pathological conditions), is proof that the thoracic diaphragm is always pressing downwards upon the epigastric viscera, and shows how erroneous is Matthews Duncan's hypothesis.

This pressure, which prevails in the abdomino-pelvic cavity, is the resultant of the weight of the abdomino-pelvic contents, i.e. of the viscera collectively with their contents, adventitious parts and tissue fluids (blood and lymph) ; plus the effect of the contraction of the muscular walls of the containing cavity which compress this *visceral mass*. Its rises are due entirely to the increased contraction of these walls, especially to that of the abdominal wall and diaphragm, because of the range of movement of which these are capable. Whilst there is difficulty at first in realising that the thoracic diaphragm is continuously pressing downwards upon the epigastric viscera, so great is our prejudice in favour of the opposite view, a little consideration as regards the disposition of the abdominal wall muscles will convince one that they act in the way

* These measurements of the rectal pressure during straining are of course those found under experimental conditions—i.e. with the patient lying on the side: it is probable they are much higher under natural conditions, when the crouching position is assumed.

† According to Keith, the pressure in the stomach in the erect posture at ease is 6 to 12 mm. Hg. [51].

supposed. The fibres of the transversales, at least, can have but one effect—that of compressing the abdominal viscera. That the diaphragm acts in a concerted way with this, for the same end, is shown by many considerations [74], besides the positive pressure found in the stomach; and to it we shall return.

It is not easy to apportion the value to each of these two factors in the production of the intra-abdomino-pelvic pressure. At first sight, gravity seems the more potent force: thus all the earlier observers thought the visceral weight by itself accounted for the pressure values found. Weisker [103] even thought the respiratory excursions of the rectal pressure are due to the variations in height of the visceral column (i.e. the height of the column of viscera from the anus to the diaphragm), rather than the result of the variations in the contraction of the muscular walls of the abdomen and pelvis which occur with respiration. Weisker's opinion, however, is shown to be erroneous by the observation that with abdominal respiration, on expiration the rectal pressure falls as the diaphragm ascends and the height of the visceral column is increased; whilst on inspiration, as the diaphragm descends, the visceral column is reduced and the rectal pressure rises.* And many other considerations show that in the normal person at least, gravity is of secondary importance. There is no doubt that the effect of the weight of the visceral mass must be appreciable, perhaps considerable; but there is also no doubt that the muscular parietes of the abdomino-pelvic cavity largely participate, even during resting conditions, as during posture, in the production of the pressure found in the pelvis. Even

* In practice, one sees that on inspiration there is an initial rise followed by a fall. This is explained by the late participation of the thorax in the act. Thoracic inspiration causes a fall in rectal pressure—due to the expansion of the thorax and extension of the spine.

though the pressure in the rectum found in the erect posture could, from the physical point of view, be attributed to the effect of gravity alone, it is certain the same effect could be caused by the combined reactions of the muscular walls of the abdomino-pelvic cavity, even though the effect of gravity could be eliminated. In the dorsal decubitus, the pressure of 10 to 14 mm. Hg. can hardly be due to gravity; and the same objection applies to the pressure found in the lateral position (6 to 8 mm. Hg.). That these muscular walls participate in violent movements and during activity is obvious: it must be admitted they are entirely responsible for the great rises of internal pressure associated with these conditions.

It is this pressure, with its never-ceasing variations, which has caused the descent of infantile abdominal organs into the pelvis and determined the responsive adaptation of the pelvis—its change in shape, the evolution of its floor, and the position of its aperture—whereby this varying pressure can be successfully withstood and an equilibrium maintained. As we have seen, in the erect posture, the aperture in the pelvic floor is situated at the lowest part of the pelvis, inviting the assumption that in prolapse it is the weight of individual organs which has caused the extrusion. This idea, we have stated, is an illusion. For if a patient suffering with prolapse bears down when in the lateral position, after the prolapsed parts have been replaced, the extrusion can be seen to recur. Here gravity can have nothing to do with the displacement. On the other hand, Matthews Duncan has observed that a prolapse can be reproduced by straining in the lateral position, when the assumption of the erect posture *per se* is ineffective [24]. Such cases are not rare. In them it is only when the patient begins to

move and to do work, to cough or to strain, that the prolapse recurs. The increase of pressure produced by rising from the lateral to the erect posture is insufficient in these cases to cause the extrusion. For this to occur an even greater pressure is necessary, such as actual measurements show is produced by bodily movements, especially those associated with manual labour, or by cough, and straining as for defæcation. Such considerations alone serve to show that gravity is not the factor in the causation of prolapse.

But the position of the pelvic floor aperture, in its relation to the direction of visceral impactions, is much more significant. For, owing to the obliquity of the pelvis and the recession of its cavity from that of the abdomen, caused by the lumbar lordosis and rendered conspicuous by the promontory of the sacrum, when great and sudden rises of pressure occur within the abdomen, as, for example, from cough, the visceral thrust from above is reflected by the lower abdominal wall (hypogastrium) into the pelvis, and is directed downwards and backwards, so that it impinges on the superficies of the pelvic cavity, far behind the aperture. Thus, the pelvic floor aperture, in virtue of its anterior position, is out of reach of the direct visceral thrust. In the erect posture, such pressure-waves only reach the aperture itself by further reflection and diffusion, not by direct transmission ; and, owing to the plasticity or viscosity of the visceral mass, they tend to weaken by diffusion. Thus, in spite of its dependent situation in the pelvis, the aperture in reality is in the most safe, most advantageous position.

That the lower abdominal wall plays the part stated in the transmission of pressure impulses from the abdomen into the pelvis, is evidenced by its marked bulging on coughing. Owing to the lumbar lordosis and the better

development of the thoracic diaphragm posteriorly (crura) than anteriorly (sternal fibres), and the direction of its costal fibres (downwards and forwards),* the effect of a suddenly produced contraction of the diaphragm is to direct the visceral downthrust somewhat forward towards the hypogastrium. It is clear the resistance of this part of the abdominal wall to this impact (cough) from above must cause a reflection of the pressure wave into the pelvis in a downward and posterior direction. It cannot help doing so. So also, the marked concavity of the pelvis posteriorly, i.e. of the sacrum, is evidence of this posterior impaction. How else could its concavity have been caused? We have but to compare the shape of the pelvis of the adult with that of a full-term fœtus to be sure of this impaction posteriorly. For with every movement of the child, with every scream and laugh, with every change in posture, and especially with the

* The crural fibres are longest of all; the costal fibres, long behind, become progressively shorter from behind forwards; the sternal fibres are shortest of all.

FIG. 12.—THE ABDOMINO-PELVIC CAVITY IN THE MESIAL SAGITTAL PLANE, SHOWING THE DIRECTION OF VISCERAL IMPACTIONS PRODUCED BY SUDDEN INCREASED CONTRACTION OF THE DIAPHRAGM AND THEIR DEFLECTION BY THE RESISTANCE OF THE LOWER ABDOMINAL WALL INTO THE PELVIS POSTERIORLY.

From which it appears that the Obturator closing in the bony pelvic outlet must be a Pressure-Resisting Mechanism; and, how it is that the Pelvic Floor Aperture, though placed in the most dependent part of the Pelvis, is in the most advantageous position. Thus, too, the bulging of the pelvic cavity posteriorly is explained.

c.t.d. = central tendon of diaphragm.
st.f.d. = sternal fibres.
cos.f.d. = costal fibres.
cr.f.d. = crural fibres (indicated by interrupted thick black lines).
d.v.i. = direction of visceral impactions.
d.r.i. = direction of visceral impactions after reflection by anterior abdominal wall.
a.c.r. = ano-coccygeal raphe.
sym.pu. = symphysis pubis.
p.r. = pubo-rectalis (the two arms of which are in the horizontal plane).
pl.fl.ap. = pelvic floor aperture.

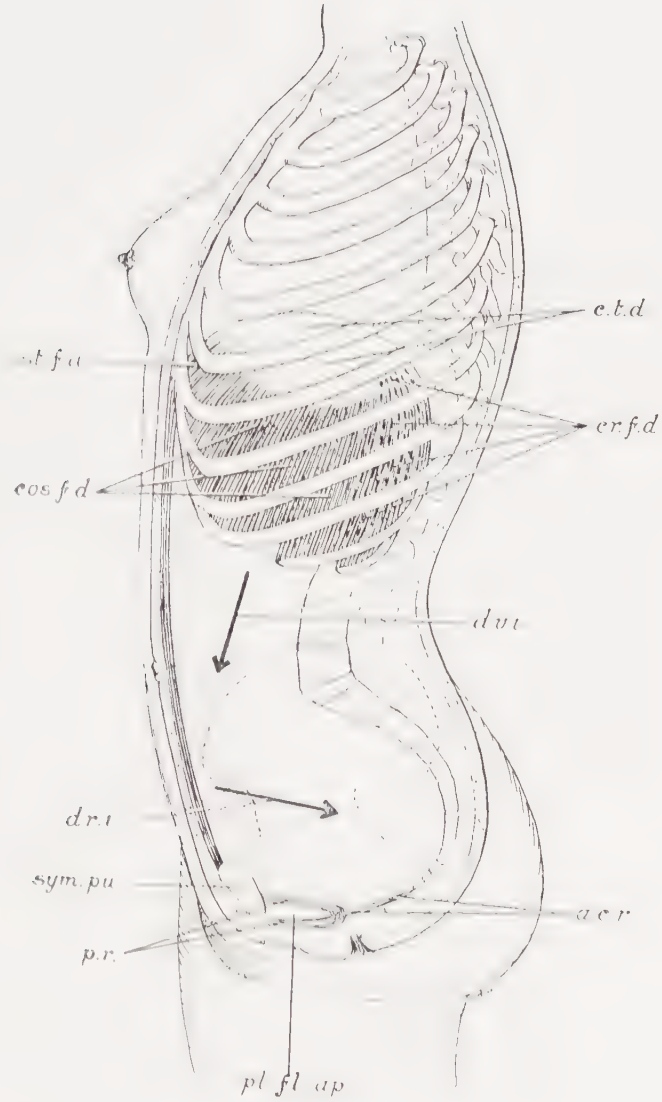

FIG. 12. *For description see opposite page*

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variations of pressure caused by walking, running, and playing, these visceral *impactions* are constantly recurring. It is the sacrum which bears the brunt of them. Thus, we perceive how the pelvic floor musculature itself is safeguarded from the effect, or rather from the full effect, of sudden visceral thrusts. Much more, then, is the dangerous region of the aperture preserved from them.

It is, as clinical experience teaches, these sudden and great excursions of pressure arising within the abdomen of the adult, in whom the pelvic floor has been damaged by childbirth or weakened by other conditions, which are so liable to cause extrusion of the pelvic viscera through the pelvic floor aperture. Obviously, if the aperture were situated posteriorly, or if it extended as a longitudinal slit from the symphysis to the coccyx, as in carnivorous pronograde mammals, such as the dog, extrusion of the viscera would much more easily occur. But these diffusion waves reach the aperture: the muscle guarding it, however, contracts to meet them, and thus in the normal prevents the tendency to visceral escape. Each time the patient coughs, the pubo-rectalis muscle can be felt to contract. The presence of this sphincter, the constriction of the obturator, and the physical nature of the pelvic visceral tissues, enable the *average* pressure to which the viscera are exposed, as also the waves of increased pressure weakened by diffusion, to be successfully met.

CHAPTER V

THE POSITION OF THE UTERUS: THE INFLUENCE OF SURROUNDING VISCERAL PARTS

1. The Pelvic Cavity and the Pelvic Viscera

But whilst the existence of this visceral pressure has been generally neglected, undue importance has been attached to the effect of gravity on individual organs, that is to say, to their weight such as they measure in air: * and this coupled with the low position of the vaginal orifice and its easy dilatability, at least in multiparæ, has led to no end of difficulties and errors. It has led to the notion of the necessity for the suspension of the cervix by ligaments, which the more one examines, the more untenable it is seen to be.

For those who believe in the suspension of the cervix by ligaments have neglected other factors, especially the influence of other viscera within the pelvis on the uterine position. They take no account of the fact that the pelvis is full of viscera; that all possess volume; and that all, though deformable, are incompressible: conditions which clearly must affect the position of any one organ, for the capacity of the pelvis is limited, and whilst the volumes of individual viscera are constantly changing, the space occupied by any one organ cannot be occupied by another. They do not see that although the thoracic

* Apart, that is, from the fact that the other organs also possess weight and tend to gravitate downwards, and must therefore have some influence on each other, affecting the position of any one organ.

diaphragm and abdominal wall are continuously adapting themselves (as regards their positions) to the changing volume of the abdominal viscera collectively, and are always in apposition with the mass these viscera form, limiting the cavity containing them, that these musculatures are doing much more—indeed, actively compressing the viscera, causing a pressure amidst them which is transmitted to the pelvic viscera, and causes them to press against each other and against the walls and floor of the pelvic cavity; and that this pressure, with its variations, must influence the pelvic visceral positions.

That the viscera are pressed against each other is shown not only by the measurements of the pressure within accessible organs, to which we have referred; but also by the presence of facets on the visceral surfaces where opposing parts have been in apposition. Such facets are readily seen in the dead, especially when bodies have been prepared by some hardening process. But even at ordinary post-mortems, I have seen them well marked, the form of the organs being fixed by rigor mortis. The intestinal coils are frequently found faceted; possessing plane surfaces separated by ridges or even sharp edges, showing how they have been adapted to the forms of surrounding viscera and pressed against them. Even the posterior surface of the uterus may show distinct facets (which I have seen), separated by distinct ridges where the intestinal coils have been pressed against it.

It is true that during life these facets are not observed—for example, during laparotomy; but this is easily explained. For, on the one hand, the organs readily alter their shapes in response to pressure changes; and, on the other, when the abdomen is opened (as also when the belly wall musculature is relaxed by the narcosis), the pressure conditions are disturbed—the compression of the viscera by the abdominal wall and diaphragm disappears

or is much reduced. Under such conditions, the viscera tend to become spherical, or their surfaces curved; because the pressure within them—due to the tension within their component cells, to the blood pressure within their capillaries, and to the pressure caused by the visceral contents—is then greater than the pressure of the air to which they are exposed. But, under natural conditions, the viscera are pressed against each other, so that the pressure between them becomes equal to the pressure within them, or approximately equal. Such at least must occur, except when individual organs are greatly distended by contents, or when their walls contract: in which cases these organs become, or tend to become, spherical. But, apart from these cases, opposing surfaces become flattened; just as a collection of spherical cells, e.g. fat-laden connective tissue cells, when pressed together become polyhedral, but when separated from adjacent cells by teasing, assume the spherical form.

From the conditions found in the dead, we must believe such facets occur in the living, and are as transient as the conditions which produce them: that with the adjustment of individual organs to variations of pressure and of volume in themselves and adjacent organs, which are constantly occurring, and the changes in shape which these variations induce, the facets constantly alter; that they become more or less obliterated during activity or distension of individual organs, whilst they must become simultaneously exaggerated on surrounding, inactive, and more or less empty organs.

But these facets, evidence as they are of a pressure relationship between adjacent viscera, do not seem to have attracted the attention of writers discussing the statics of the pelvic viscera. And, unhappily, in judging of the factors which maintain the uterus at its level in the

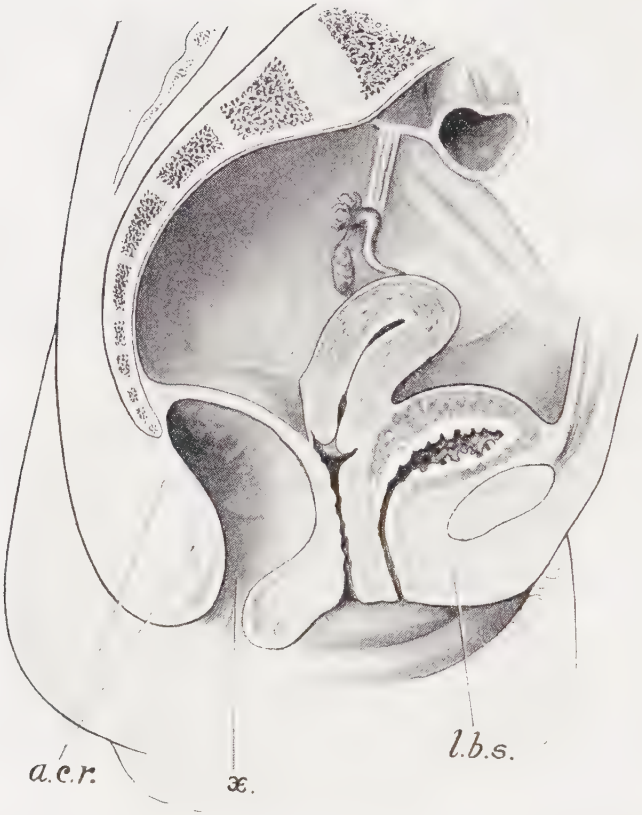

FIG. 13.—A SECTION OF THE PELVIS AND VISCERA IN THE MESIAL ANTERO-POSTERIOR PLANE, IN WHICH THE BLADDER IS REPRESENTED AS AN OVOID ORGAN, AND THE UTERUS AND VAGINA MORE OR LESS VERTICAL: IN WHICH THE IDEA THAT ALL THESE VISCERA ARE PRESSED AGAINST EACH OTHER AND AGAINST THE PELVIC WALLS AND FLOOR IS CONSPICUOUSLY ABSENT.

(After Cameron).

For remarks on ovoid form of bladder in the adult see Text and Footnote, p. 137.

Note the nearly vertical position of the ano-coccygeal raphe (a.c.r.); and the distance of the upper part of the anal canal (x) from the lower border of the symphysis (l.b.s.).

[*To face page* 59

pelvis, even the normal position of the uterus as well as its relations to adjacent viscera, appears by some to have been lost sight of. It is suggested, at least by diagrams (Fig. 13)—the suggestion being favourable to the connective tissue hypothesis—that the uterus and vagina are vertical in the adult, standing erect as a column over the pelvic floor aperture—represented as a patent gap between the levators—as though the uterus rested on the vaginal summit, as used to be believed; or, as though the uterus remains poised in this position by the connective tissue attachments of the cervix and vagina to the lateral pelvic walls—the suggestion being that these attachments prevent the cervix slipping down the vagina in virtue of its weight. As a matter of fact, we know that in the adult, the long axes of the uterus and vagina, instead of being vertical and in the same straight line with each other, are very obliquely inclined in opposite directions—both to each other and to the pelvic floor—as though they had been pressed from above downwards, with the other pelvic viscera, on to the pelvic floor (Fig. 6, p. 24); that when the pelvic floor is in its normal position and the rectum is empty, the vagina passes in a direction very obliquely backwards, much more backwards than upwards*; and that when the bladder is also empty, the uterine axis passes from the cervix to the fundus much more forwards than upwards, often, indeed, being horizontal, and in some cases actually passing forwards and somewhat downwards, so that the fundus is on a lower level than the cervix—positions which are not favourable to the connective tissue hypothesis.

And not only the positions but the forms of the pelvic viscera suggest this pressure from above, which clearly posits a supporting superficies below, successfully opposing this downthrust. Thus, the vagina, in its intra-pelvic

* The body being in the erect posture.

part, is spread out from side to side, being flattened from above downwards, so that its superior (anterior) and inferior (posterior) walls are in apposition. But in its extra-pelvic part (in that part of its course in which it traverses the superficial subcutaneous tissues from the level of the pelvic floor to its orifice at the hymen), it is flattened from side to side by the superficial sphincteric muscles, which pass backwards laterally by it to the perineal body. And within the pelvic floor aperture, in company with the gut and the urethra, it is crumpled and constricted. Here, owing to the adhesion of its lateral walls to the muscle forming the lateral boundaries of the aperture, and to the presence of the gut behind and the urethra in front, which bulge its posterior and anterior walls respectively forwards and backwards, forming the columns of the vagina, its lumen is H-shaped on transverse section—the result, clearly, of constriction. And since at this place it is pulled forwards with the rectum by the sphincter muscle of the aperture, so that its anterior wall is in close relation to the pubic arch, its long axis is not in the same straight line with that of its intra-pelvic part ; but the two form a curve with the convexity directed forwards and upwards, apposed to the pubic arch, similar to, but less marked than the perineal curvature of the gut. But its general direction is such that as it pierces the pelvic floor, its long axis cuts the median superficies of the pelvic floor very obliquely. Within the pelvis, it lies almost parallel with the pelvic floor, separated from it by the terminal part of the rectum and some connective tissue.

The terminal part of the rectum lies between the vagina and pelvic floor amidst fatty connective tissue ; and when empty, like the vagina, is flattened from above downwards on to the pelvic floor. In the erect posture, it passes much more forwards than downwards; and before

it turns downwards and backwards through the pelvic floor aperture to become the anal canal, it approaches to within quite a small distance of the pubic arch—being separated from it only by the vagina and urethra. Emphasis is to be laid on this near approach of the rectum to the anterior bony limit of the pelvic floor aperture; it is a most important relationship from our point of view, and erroneous impressions about it are current. In many modern text-books on anatomy, it is represented at quite a distance from the bone (as in Fig. 2, p. 17); but if a finger be passed into the anal canal in the normal* and directed anteriorly, the pubic arch will be come upon almost immediately—the ease with which the bone can be felt, which is due to the nearness of the perineal flexure of the gut to the pubic arch, will cause very great surprise to any one making this examination for the first time. This dragging forwards of the rectum and formation of its perineal curvature is due, as we have said, to the activity of the pubo-rectalis muscle forming the lateral and posterior boundaries of the pelvic floor aperture.

As to the uterus, it lies on the bladder: its fundus pointing forwards in anteversion and directed a little to the right (Boivin and Duges); its cervix pointing backwards and directed a little to the left—a position maintained by intestinal coils above, which press downwards upon its postero-superior surface. The cervix abuts upon the lower part of the rectum—either medianly, or to one or other side of it: it is separated from the rectum by the posterior vaginal wall and Douglas's pouch, a duplication of peritoneum which dips down just beyond the cervico-vaginal junction, and the opposing surfaces of which are usually in apposition. Thus, the cervix can easily be felt by a finger in the rectum; and obviously, is not far

* Choose a robust healthy virgin or nullipara.

removed from the part of the pelvic floor in this region. In spite of the variability of its position, the cervix may be said to be placed on a level with the ischial spines and more or less midway between them ; and, in sagittal plane, on a level with the superior border of the symphysis, a little beyond the mid-point of a horizontal line drawn backwards from it, which line impinges posteriorly on the sacrum inferiorly (lower part of the fifth sacral vertebra), Thus, whilst the cervix is relatively close to the pelvic floor. it is some distance from the pelvic wall behind. But in front, when the bladder is empty, the fundus comes into much closer relationship with the limiting wall (pubes)—a layer of visceral connective tissue, the upper duplication of the bladder and the peritoneum alone intervening. Thus, it is that, the cervix being fixed by two fingers in the vagina and the lower abdominal wall being depressed towards or into the sloping brim by the other hand, the fundus of the uterus can readily be discerned. The body of the uterus is wider from side to side than from before backwards (or from above downwards) : and whilst its superior (posterior) surface is more rounded, its inferior (anterior) is nearly flat, suggesting it too has been flattened from above downwards, like the vagina and other pelvic viscera. And Astruc [5] in 1740 attributed the flatness of the uterus to the pressure of the bladder and rectum by which he thought the organ was kept in place.

The bladder when empty is a saucer-shaped organ, into the upper concavity of which the body of the uterus is pressed. Like the rectum and vagina, it also is flattened from above downwards. It is spread out from before backwards and also from side to side over the superficies of the pelvic cavity to which it is related. It separates the body of the uterus from the vagina, extending from the anterior limit of the pelvic cavity backwards to the cervico-vaginal junction ; and causes the long axes of

these two parts to meet, generally, in an acute angle. And it extends laterally, beyond the uterus above it, and the vagina below it; coming into contact (except for the peritoneum) with other viscera at the sides of the uterus above; and (except for loose connective tissue) with the pelvic floor musculature below, on either side of the aperture and vagina; and at the sides where its superior and inferior walls meet it comes into relation with the lateral pelvic walls. Medianly, its lower wall or base is related to and rests upon the median part of the pubic arch, separated from it by loose connective tissue; behind this, it is related to the pelvic floor aperture, normally closed by its obturator (urethra, vagina, anal canal and connective tissue); posteriorly, it is related to the vagina. The existence of the vagina and rectum beneath this part of the bladder base must naturally elevate it, sufficiently explaining the existence within the bladder of the para-trigonal fossæ—shallow pouches on either side of the trigone.

Thus, if we take a pelvis and divide it by a coronal section passing through the diameter of the brim—not at right angles to the brim, but more obliquely (i.e. in a plane nearer the horizontal)—we see that the uterus and bladder occupy the anterior and dependent part of the cavity, whilst the intestines occupy the parts above and behind. The empty bladder, with the uterus above it (also horizontal), is pressed downwards on to the parts below, and comes into close relation with the anterior and antero-lateral pelvic walls and with the anterior parts of the pelvic floor and the aperture between them, adequately explaining the saucer-shaped form of the lower organ—a relationship only disturbed by the passage forwards of the vagina and rectum to gain the pelvic floor aperture. Thus, when the pelvic floor aperture is dilated, as by straining, the anterior vaginal wall, with the part of the bladder

above it, and (when the rectum is distended) the posterior vaginal wall with the distended rectum behind it stretch over the aperture and are bulged into the aperture; but when the aperture is similarly dilated during defæcation, the passage of the fæcal mass, by enlarging the anal canal, fills up the aperture and prevents such bulging of the parts above. To a much greater extent is the pelvic floor aperture dilated and blocked by the fœtus during parturition.

The idea that the body of the uterus is supported on the vagina as on a column, that the two are vertically placed, is clearly erroneous. No one believes this is so; but, strange to say, the idea is still displayed. It is obvious the position of the uterine body, when anteverted, depends on that of the bladder; for it varies with the volume of the bladder—the uterus is raised and displaced backwards when the bladder is distended, and pressed again into a horizontal position when the bladder becomes empty. It is clear that the forces which support the fundus and body of the uterus at its level are those which maintain the bladder in place: but the structures imputed to do this, are incapable of it. The urachus, the ureters, the superior vesical arteries, the connection of the bladder with the prostate in the male (and presumably with the cervix in the female), the so-called anterior and lateral true ligaments of the bladder, have all been pressed into this service [93]. But even these all acting together have been unable to prevent the ontogenetic descent of the bladder—its descent, that is, from the abdomen into the pelvis which occurs with growth; nor in the adult are they capable of preventing its further displacement. Even though the condensations of the connective tissue beneath the bladder and those that exist about the vesical blood-vessels, lymphatics and nerves, magnified into ligaments, only occur with growth; these in the adult are inconspicuous structures. According to Martin, the pubo-vesical ligaments are the most im-

portant means for holding the bladder in position: they are rendered effective, since besides being attached to the pubes in front, they are connected posteriorly with the so-called ligaments of the cervix through which they gain attachment to other parts of the pelvic walls [61]. But Halban and Tandler say the pubo-vesico-uterine ligaments are such poorly developed structures that it is impossible to attribute much value to them in this respect; and in recent text-books of importance I can find no mention of them. Whilst some writers believe the lateral ligaments of the cervix, the cardinal ligaments, or the condensations about the uterine blood-vessels are the most important of the so-called cervical ligaments; Eden and Lockyer, from their operative experience, are of the opinion that the utero-sacral ligaments—the variable amounts of firm fibrous tissue with a little plain muscle contained within the utero-sacral folds—are "firmer and stronger than the other 'ligaments'" [26]. Thus, we can truly say, that the connective tissue in which the bladder is embedded, whilst it contains some strands of thickening, contains no such thickenings as that of the cervix possesses; and this in spite of the much greater bladder size and weight when contents are present: but its conspicuous feature is that it is loose in texture and in obese people fatty. And if it be believed that the cervical ligaments (e.g. the utero-sacral) are the cause of the position of the cervix and of the uterine body, since the position of the uterine body varies with and depends on that of the bladder, to these also must be attributed the posterior duplication of the bladder—and thus the shape of the bladder—and also the acute inclination of the long axes of the uterus and vagina to each other—a most unlikely hypothesis. But the positions and forms of all these viscera become intelligible when we recognise they are pressed downwards from above on to the pelvic floor.

2. The Factors at Play in the Maintenance of the Pelvic Visceral Positions

Just as it has been assumed that the connective tissue attachments of the cervix directly support the weight of the uterine body, in spite of the anterior position of its fundus and the saucer-shaped form of the bladder into the concavity of which the uterine body is pressed, so it has been assumed that the position of the cervix determines the position of the body of the uterus (Schultze) [90], because the cervix is embedded in connective tissue and is relatively less mobile than the fundus. But when we think of the difference in size between the uterine body and cervix, it is plain that forces of equal magnitude, acting on both simultaneously, will affect the position of the fundus more easily than that of the cervix. So the view, that in virtue of the relative rigidity of the uterus, the posterior position of the cervix is due to the anterior position of the fundus is overlooked. Clearly, the pressure from the abdomen on the broad posterior surface of the uterine body, which when the bladder is empty is more superior than posterior, tends to maintain the cervix in its posterior position. So also, we know, that when the bladder is distended and the uterus more or quite vertical, the bladder itself causes the cervix to become more posterior still. And if the uterus is not rigid, or if its body has been removed by a sub-total hysterectomy, this pressure from the abdomen upon the bladder, whatever its state of distension, will act in the same way on the cervix. Even if we leave out of account the pressure from above, the effect of gravity (in the erect posture), which of course affects all the viscera simultaneously, will tend to maintain the cervix posterior by pulling or attracting the larger and heavier uterine body downwards: and when the bladder contains urine and

the fundus of the uterus is raised, gravity acting on the bladder and its contents alone, will have a similar effect.

Such considerations show that the position of the cervix does not depend on the effect of gravity on the cervix; but they show equally clearly that the posterior position of the cervix depends on the existence of visceral parts in front of it—on the corpus uteri and on the bladder when the bladder is empty, and on the distended bladder alone when the bladder is distended—all of which are subject to the same physical laws as the cervix. Yet gravity, presumably, is not without effect on the cervix, for the cervix possesses some small mass; but, obviously, this effect is obliterated or compensated for by the effect of gravity on the much larger and weightier uterine body and empty bladder, or on the greatly increased bladder mass when the bladder is distended. This obliteration of the effect of the cervical weight, it may be argued, is shown by the change in the position of the cervix in response to a change in mass of the parts in front of it. So, if gravity determines the position of the cervix, it does so indirectly by acting on these other parts.

But besides their mass and weight, the uterine body and bladder possess volume also: they occupy a definite space in the pelvis—a much more important physical relationship. For besides gravity, they are subject to other forces in the pelvis. On the one hand, they are pressed downwards by the compression of the viscera above, plus the collective weight of these viscera (in so far as this may be effective); and, on the other hand, they are supported by the pelvic floor. This compression of the bladder between the pressure above and the resisting pressure below, clearly maintains the cervix in its posterior position. It does so in virtue of the volume and incompressibility of the viscus. For the compression

to which the bladder is subjected is so considerable as to maintain a flattening of it from above downwards, not only from side to side, but from before backwards, so that the bladder lumen extends from the pubes to the cervix. Gravity acting on the bladder alone could not determine this: nor, when we think of the spherical shape of the bladder in the new-born child, can we believe that gravity acting on the entire visceral mass would or could —in the course of ontogeny—have caused this shape.

The cervix is not only displaced farther backwards by distension of the bladder, but also by increase in volume of the uterus, as in early pregnancy. In this condition, owing to the softness of the uterine tissues, the cervix is flexed on the uterine body, often acutely—the normal anteflexion is increased. And, besides, the cervix is considerably raised, becoming more difficult to reach *per vaginam*. This posterior dislocation of the cervix, the result of distension of the bladder or of enlargement of the uterus, shows the resistant effect of the anterior bony wall of the pelvis, which limits anteriorly the space these organs occupy. Similarly, the elevation of the cervix and the increased anteflexion in pregnancy, show the influence of the visceral parts behind and below the cervix, and the effect of the resisting superficies of the pelvic cavity against which these parts abut—the sacrum and the pelvic floor.

For the position of the cervix depends, besides on the volume of the parts in front of it and on the force with which these are pressed downwards, upon the existence of visceral parts behind and below it, and varies with their volume. As the rectum is being distended with fæces, it first necessarily pushes the cervix and the upper part of the vagina, and with these the ante-posited bladder, forwards; and then, as the terminal part of the rectum, which is placed below the cervix and this part of the vagina, distends, it pushes them upwards. For the sacrum is immobile; and

the pelvic floor, even on marked straining (under experimental conditions), only descends about half an inch, and this only anteriorly in the region of its aperture [73]. Since the visceral parts in front of the rectum abut anteriorly and laterally on the bony pelvic walls, the anterior movement caused by distension of the rectum must result in their elevation. But besides this, changes in form of the viscera occur. If the uterus be flexible, its anteflexion occurs or is increased; for the pressure from above acts on the fundus: but if rigid, the uterus is bodily displaced (Schultze) [90], overcoming this pressure. And whilst the upper wall of the bladder bulges, elevating the uterus, its basal part becomes shortened, perhaps crumpled, from behind forwards—the shortening depending on the elasticity of its tissues and the advance of the cervix. If we imagine the bladder to be so distended by the pressure of urine within it, that its antero-posterior diameter cannot be much flattened at the expense of its vertical, and shortening of its base cannot occur, the uterus, which is then vertical, will be bodily elevated—the cervix slipping upwards between the bladder and the enlarging rectum, as it obviously must do to allow the fæcal mass to pass downwards; and, when this is directed forwards and enters the terminal part of the gut, the bladder itself must be bodily lifted to afford the necessary room.

The elevation of these visceral parts clearly depends on the position of the pelvic floor: since the pelvic floor descends with defæcation, the elevation is correspondingly reduced. When the rectum empties, a reverse movement occurs: the pelvic floor rises to its ordinary resting position, and the pressure from above presses the uterus and bladder back into their former positions. In the same way, too, one cannot but believe that variations in the volume of the vagina must alter the position of the cervix and other viscera (e.g. bladder). For example, the

growth of a cyst in the vaginal wall, if of any size, or a hypertrophic elongation of the cervix, must cause changes in the positions of surrounding parts, for the most part elevating them.

Just as the position of the uterus, in the non-pregnant state, depends on the volumes of the other pelvic viscera and varies with these, and on the pressure imparted to all by the adjacent abdominal organs; so in pregnancy, the uterus, as it enlarges, not only causes changes in the positions of the surrounding viscera and in the positions of the muscular walls of the abdomino-pelvic cavity, but the incompressibility of the former and the resistance of the latter react upon the uterus itself, causing changes in its shape and determining or affecting its position. In the early stages, it is obvious that the rectum, in virtue of its volume and incompressibility, with the connective tissue about it and the pelvic wall behind, plays a part in causing the increased anteflexion of the uterus. On the one hand, the cervix is pushed backwards, because the expansion anteriorly of the anteverted uterus is obstructed by the bladder supported by the pubes; and, on the other, the upper part of the vagina with the vaginal portion of the cervix are prevented from receding beyond a certain limit by the rectum, in turn supported by the posterior pelvic wall. Since the pelvic floor usually remains in its normal position, or is not appreciably altered—at least at this time—and the visceral parts below the uterus do not change in volume, but the space they occupy is encroached on by the uterus as it grows, the reason of the simultaneous elevation of the cervix becomes plain.

And, with its further expansion, further changes occur. The viscera above it, the pelvic colon and other intestinal coils, are displaced upwards or to the sides. At first it produces a distension of the lower, and then of the upper

abdomen; and finally, a distension of the thoracic base by pressing the epigastric viscera upwards against its roof and walls. The effect in the pelvis, if not so extensive, is no less marked. The position of the bladder especially is altered: its lower posterior part is more compressed than ever from above downwards on the parts between it and the pelvic floor, whilst its anterior part is similarly more compressed from behind forwards against the pubes. Consequently, it becomes more spread out, both from behind forwards and from side to side. This is easily demonstrated with the sound. Since the fœtal parts in the lower uterine segment are felt so easily through the anterior vaginal wall, it is a matter of surprise to find that the bladder lumen still extends as far backwards as the cervix: for one cannot perceive the bladder walls by the finger, but the sound readily demonstrates their limit. And just as the cervix is placed more posteriorly in the pelvis, so the duplication of the bladder posteriorly has been similarly displaced backwards. So also one finds, by means of the sound, that the lateral and superior duplications of the bladder have been extended. In the latter case, the point of the sound can be easily felt through the abdominal wall: in one case I examined, it was discovered even though the bladder was empty, and without using any force, at a distance of $2\frac{1}{2}$ inches above the upper border of the pubic symphysis [70].

According to Schultze [90], some little descent of the pelvic floor occurs in pregnancy; but this is so small that from our point of view it may be neglected. Sometimes, however, a very considerable descent occurs with a corresponding lowered position of the pelvic viscera, especially of the cervix and bladder; and often some prolapse—of the anterior vaginal wall and bladder—is present. Notably is this the case when corsets have been worn with the object of preventing the normal distension of the abdomen, for the purpose of hiding the condition of pregnancy—not

infrequently done by single women who have become pregnant. This was demonstrated to me in a very striking manner at the time I was studying the superficies of the pelvic floor and the pelvic floor aperture by measurements. A primagravida, twenty-two years old, single, and seven months pregnant, was examined on October 15th, 1908, shortly after being admitted to a maternity home. On that day, I found the distance along the superficies of the pelvic floor in the median plane, from a point midway between the two posterior superior iliac spines and the mid-point of the upper border of the symphysis pubis, to be $14\frac{1}{2}$ inches. But on December 10th, 1908—two days, as it happened, before delivery—a measurement beween the same two points gave the distance as $12\frac{1}{4}$ inches. Even allowing for errors in making the measurements, it is clear that a considerable diminution had occurred. This was explained by the patient's statement that she had worn her corsets "very tight" before entering the home: whilst, during her stay, she had not been allowed to wear them at all, and moreover had spent a good deal of her time in bed.

But besides the volume of surrounding viscera and the resistant superficies of the pelvic cavity, there is another element in the pelvis which plays a part in the determination of the uterine position. This is the visceral connective tissue, in which the viscera with their peritoneal sac, blood-vessels and other parts, are all embedded. It is obvious that this connective tissue, especially that situated between the viscera and the pelvic walls and floor, like the viscera themselves, in virtue of its *volume* and incompressibility, plays a part in the determination of the uterine position: that when the volume of this connective tissue is increased, by a deposit of fat in its cells—which occurs peripherally, around the bladder, vagina and uterus collectively, and

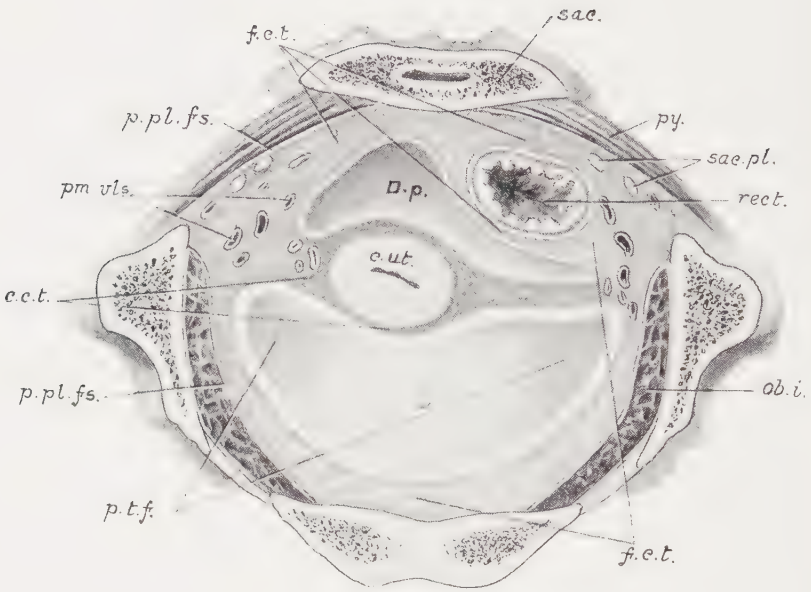

FIG. 14.—A HORIZONTAL SECTION THROUGH THE PELVIS TO SHOW THE MAGNITUDE OF THE VISCERAL CONNECTIVE TISSUE, AND ITS RELATIONS TO THE PELVIC VISCERA AND TO THE WALLS OF THE PELVIC CAVITY.

(After Martin.)

Note that the pyriformis muscles close in the space on each side between the sacrum and the ossa innominata (great sacro-sciatic foramina); and that these muscles are lined internally by parietal pelvic fascia which pass outwards with the respective muscle (and with blood-vessels and nerves) through these foramina, and do not become continuous with the periosteum of the bone in front of it.

py. = pyriformis.
sac.pl. = sacral plexus.
p.pl.fs. = parietal pelvic fascia.
pm.vls. = parametric blood-vessels.
f.c.t. = fat-laden connective tissue.
c.c.t. = condensed connective tissue.
ob.i. = obturator internus, lined by parietal pelvic fascia.

[*To face page* 73

around the rectum*—the cervix will be situated at a

* The truth of this statement that fat occurs in the visceral connective tissue in the pelvis, beneath and low down at the sides of the pelvic viscera, between them and the pelvic floor, may be doubted. I do not know whether its occurrence is generally recognised, and I have found no reference to it in recent books; but one reads, on the contrary, of the loose fatless connective tissue, with abundant blood-vessels and lymphatics, which surrounds the lower part of the uterus and the upper part of the vagina, spoken of as the parametria, and which suggests there is no fat in the pelvis.

In older writings, however, one comes upon the statement that fat exists in the pelvis. Thus, Smellie [92] says the vagina on its outside is "covered with a thick adipose membrane"; and Levret [57] says all the nerves, veins and arteries which are distributed in the pelvis are maintained and supported by a cellular or fatty tissue, which is flaccid and soft. Veit [102], in 1877, referred to a demonstration by Hoffmann of sections of a pelvis from a woman dead shortly before confinement, in which, in spite of the well-developed fat in the subcutaneous tissues, no fat was present in the pelvis; in contrast to the plentiful perivaginal fat in an otherwise only moderately well developed girl.

To satisfy myself on this point I examined the connective tissue of the pelvis during post-mortem examinations when Pathologist at the Hospital for Women, Soho Square. I found if the collective pelvic viscera are removed in one piece from a fat corpse—one would not expect to find fat anywhere in a thin one—fat is present in the pelvic visceral connective tissue.

Such a specimen, clearly showing fat, was in my possession. I removed it from a patient who was admitted after an abortion to the Hospital for Women, Soho Square, and who died from sepsis. The mass when removed was hardened in formalin, and then cut antero-posteriorly in the median plane. In the plane of the section, a thin yellow layer of fat is seen in the connective tissue in front of the rectum, separating it from the peritoneum forming the posterior wall of Douglas' pouch, and passing downwards between the rectum and vagina. This fatty layer is continuous with a layer of fat surrounding the rectum. It can be traced forwards by the side of the vagina to the lateral aspect of the bladder and inwards beneath the bladder anteriorly to the median line. It extends upwards at the sides, away from the uterus, beneath the peritoneum lining the lateral wall of the pelvis; in front, it extends upwards in front of the bladder; whilst behind, it similarly extends upwards around the rectum; to reach on all sides and to become continuous with the sub-peritoneal fatty tissue in the abdomen. Whilst surrounding the rectum, being directly adjacent to its walls on all sides, it does not invest the uterus, nor does it pass inwards in the connective tissue between the cervix and the bladder. On making a horizontal

higher level in the pelvis than when this connective tissue contains no fat, and thus must possess a lesser volume. Matthews Duncan commented on the relatively high position of the uterus in individuals in whom there is a good deposit of fat beneath the skin of the anterior abdominal wall. He thought this influenced, in some strange way, the retentive power of the abdomen*: but a deposit of fat in the subcutaneous tissues does not occur apart from a similar deposit in the visceral connective tissue.† So also we know that ptosis of the abdominal organs is much more readily induced, and much more often seen, in thin than in corpulent persons.

section through the cervix and adjacent tissue, the fat is seen to form a peripheral layer, investing not the cervix but the dense connective tissue about the cervix. The so-called parametric tissue is to the naked eye free from fat.

Fat sometimes occurs in the broad ligaments. I have several times seen it about the Fallopian tubes removed at operation. Other specimens, showing the presence of fat about the pelvic viscera, have also passed through my hands. In the case of a uterus and adjacent part of the vagina removed by Wertheim's operation for carcinoma of the cervix and infiltrating the vagina, section through the diseased mass showed it abutting upon fatty tissue—confirmed by microscopic examination.[a] So also in the case of a rectum, removed for a carcinoma just within the anal canal. An extensive operation was done, the skin, ischio-rectal fat, part of the levator ani and eight or ten inches of the rectum were removed in one piece.[b] Fat was everywhere present, investing the gut, extending right down to the levator ani muscle, which separated it from the large fatty deposit around the anal canal (ischio-rectal fat).

[a] The patient was under the care of Dr. Simson.

[b] The patient was under the care of Mr. Drew.

* "Fat in great quantity in the anterior abdominal wall generally prevents prolapse, increasing the retentive power of the abdomen."[24] So also Levret, in a note on an aphorism of Mauriceau, says there are few women afflicted with descent of the uterus, who, when radically cured by whatever means, can do without the pessary; and when such patients are so fortunate as to be able eventually to dispense with this support, it is only because, from being very thin, they have become fat whilst using the pessary."[58]

† Except in advanced or even mid-pregnancy.

A third factor, just to be noticed, is the volume of blood in the vessels, but especially in the capillaries and veins, and of lymph in the cell spaces and channels. Though the flow of blood is continuous, its volume in any one collection of capillaries and veins varies at times according to the activity of the organs, which is associated with a dilatation of arterioles and a greater blood-flow through the capillaries. Such variations clearly must affect in a minor degree the positions of the viscera. Clearly, too, the presence of tumours in the pelvis, such as ovarian cysts, etc., affects the position of surrounding viscera. The influence of these is well known and need not be discussed.

3. The Pelvic Visceral Positions and the Intra-Abdomino-Pelvic Pressure

These simple illustrations serve to show that the position of the uterus depends, on the one hand, upon the existence of the surrounding visceral parts—on their volume, on the space they occupy, and on their mutual relations; and, on the other, on the relation of these parts to the walls and floor of the pelvic cavity. This cavity has only a certain capacity; it is packed full of viscera with their contents, adventitious parts and connective tissue: its rigid walls and resistant floor contain them collectively. Since the organs, their contents, and the connective tissue possess volume and are incompressible, yet are easily deformable, it is obvious that the uterine position must depend upon the space occupied by these other parts, and must alter as their volumes change.

And, it is clear, the force which maintains their surfaces in apposition is also of importance. Each of the factors of which this force is the resultant must therefore participate. Gravity, then, is partially responsible. Gravity,

however, cannot affect one organ irrespective of others: nor can it affect one organ more than another, unless the specific gravity of this be greater than the specific gravities of surrounding organs. The weight of individual organs does tend to cause them to gravitate downwards in the pelvis; but when we consider the position of any one organ in respect to this force, we find the weights of surrounding organs cause these also to gravitate downwards and to abut upon and to press on the surfaces of the viscus considered, and thus to obliterate the effect of its weight. If gravity is to be considered in the statics of the pelvic viscera, it is the collective weight of the viscera with their contents, adventitious parts and connective tissue—in short, of the entire visceral mass—in so far as this may be transmitted to and may affect the pelvic viscera, which has to be thought of: not the effect of the weight of any one organ such as it may measure in air. If the weight of individual organs, such as they measure in air, were effective in determining the positions of the viscera, it ought to show itself in the case of the body of the non-pregnant uterus, which is so mobile. Yet the observation of Schultze [90], that when the bladder of a woman in the dorso-horizontal position is emptied, the uterus passes into anteversion in spite of the opposing effect of gravity, i.e. of its weight, shows that its weight is ineffective in influencing the uterine position.

And how difficult would it be, on this supposition, to explain the upward growth of the uterus when pregnant, which from its contents becomes so heavy! This phenomenon, with which we are so familiar that it creates no surprise and elicits no inquiry as to its cause, exercised the minds of some of the older obstetricians very considerably. Matthews Duncan referred to the difficulty Joulin had in explaining it: indeed, Joulin could not explain it. Joulin's wonderment at the upward growth

of the pregnant uterus and its invasion of the abdomen was so great that he described it as a " most remarkable " and " most inexplicable phenomenon " ; for the uterus, he said, " leaves the excavation of the pelvis and mounts into the abdominal cavity, against all the laws of physics and without any anatomical reason to explain the ascending movement " [48]. Matthews Duncan himself attributed it to the effect of the aspiration of the thorax, just as he explained in the same way the position of the non-pregnant uterus in the pelvis, and its easy mobility. He thought the negative pressure in the thorax prevails in the epigastrium ; that it extends throughout the abdomen, and reaching the pelvis, aspirates the uterus upwards. So when the uterus becomes pregnant, its body naturally rises. He argued that the base of the thorax expands as pregnancy advances, and that this causes an increase in the capacity of the abdominal cavity. Thus, he thought " the uterus, instead of forcing its way up against the influence of gravitation, is probably actively drawn up, at least to some extent." He even thought in some cases the pregnant uterus is entirely suspended by these " drawing-up forces," and is then entirely removed from solid subjacent support [22].

The difficulty these obstetricians encountered in explaining the upward growth of the uterine body when pregnant is readily accounted for by their unawareness of the pelvic floor musculature and the work that it does—a negation then prevalent amongst students of medicine. Obviously, if the pelvic outlet were not occluded by some resisting mechanism, the retention of the pelvic viscera, even in the non-pregnant, much more in the pregnant, becomes inexplicable. But when we recognise the existence of a muscular parietes closing in the pelvis inferiorly, which we can feel with our fingers in the living, and see with our eyes in the dead—a musculature similar in every

way, both in structure and function, to the abdominal wall above—how easy and readily intelligible do these phenomena become! I cannot but wonder how those gynæcologists and obstetricians, who see in the pelvic floor musculature but vestigial remains of tail-moving muscles, a view for which there is no foundation in fact, and who on this assumption reject this musculature as a functional part of the parietes of the abdomino-pelvic cavity, explain the upward growth of the uterus when pregnant. I cannot but think it must present some difficulties, and is best left alone. It cannot very well be explained by the action of the connective tissue attachments of the cervix; for the so-called cervical ligaments become soft and the fibrous strands disappear. Clearly, the loose and succulent visceral connective tissue of the pelvis, so characteristic of pregnancy, is quite inadequate to support the pregnant uterus; much less to cause its enlarging body to grow upwards, with a force sufficient to displace the viscera above it and to cause the marked distension of the abdomen and of the thoracic base, which are such familiar sights to obstetricians. On the other hand, the hypertrophied round ligaments must have just the contrary effect, tending to pull, and pulling, the uterine body downwards when they contract.

In the case of the non-pregnant uterus, it is, as we have seen, the change in volume of the surrounding viscera which so readily and easily affects its position. That it is the increase or diminution in the space of the pelvic cavity occupied, rather than the variation in visceral weight, which usually corresponds with the variation in volume, that is effective in causing the change in the uterine position, is shown by the displacement of the uterus and other parts in front of the rectum by distension of the rectum with gas, as when it is ballooned; and by the similar displacement in a contrary direction

produced by artificially distending the bladder with air. So, it is the change in volume of the uterus in pregnancy which affects not only its position, but also the positions of surrounding viscera; for whilst its mass increases, its specific gravity remains approximately the same as that of surrounding tissues. Clearly, the volume of a body, approximately incompressible, in relation to the capacity of a chamber enclosing it, is a physical feature of essential importance for the production of a pressure on or in that body: its weight, though facilitating, is of secondary importance. So, as we have urged, the effect of the contraction of the muscular walls of the abdomino-pelvic cavity, which contraction causes the compression of the whole visceral mass in respect of its volume and squeezes the pelvic part into the pelvis, is much more important for the statics of the viscera (pelvic and abdominal) than the effect of gravity. This muscular contraction, and the compression it causes, is a vital phenomenon. It persists in the dorsal decubitus, maintaining a greater pressure in the pelvis than in the epigastrium even in this position, so necessary for the blood return to the thorax which gravity could not determine; and its persistence in this position is the reason the uterus follows the bladder when the bladder is emptied by catheter. For the uterus is insensibly pressed downwards on to the bladder by the pressure to which the viscera placed above the uterus and already insinuated into the interval between its fundus and the rectum, are subjected. This visceral compression even persists in the lateral position: but in this, it is much less marked than in the dorsal decubitus. It disappears in the knee-chest and allied positions; for its necessity has gone—not only do the viscera gravitate downwards towards the epigastrium, but the venous blood flows in this direction in virtue of its weight. But, especially is this visceral compression important; because, during activity, it is

constantly changing in intensity, frequently becoming exceedingly great.

The variations in position of the uterus, both of fundus and cervix, in adaptation to variations in the volumes of the bladder and rectum which are constantly changing; its adaptation to the volume of the visceral connective tissue, which, though more constant or permanent, yet undergoes considerable variations in the life-history of the individual; and its upward growth into the abdomen when it becomes pregnant, are but the results of variations of pressure occurring locally in the pelvis produced by these changes in volume.

For, if the pressure in the empty and uncontracted bladder (and the wall of the empty bladder is usually uncontracted) be equal to the pressure with which the viscera about the bladder press upon it (and it cannot be less in ordinary positions), urine can only be caused to enter the bladder by overcoming this pressure. The urine does not trickle down the ureters, in virtue of its weight; but it is forced down by ureteric peristaltic waves, and for this reason the ureters possess well-marked muscular walls. In order that the bladder may distend in the early stages, it is but necessary that the urine should be forced into it with a pressure sufficient to overcome the pressure on the bladder walls*; and it is this force which causes the bladder to dilate, and to raise the uterus and other viscera above it, in spite of these being pressed down upon it by the pressure from above. Clearly, the displacement of these surrounding viscera tends to allow the pressure within the bladder, causing its distension, to fall—that

* Later, as the bladder becomes distended, the force with which the urine is squirted into the bladder has to overcome the elastic resistance and the recurring rhythmic contraction of the bladder walls themselves.

is to say, the pressure in the bladder does not rise as it would do if displacement of surrounding viscera was difficult or impossible. But the bladder pressure does not remain the same as before; with increase of contents, some increase of pressure also occurs.

Similarly, the rectum can only distend with fæces by these being forced into it with a pressure sufficient to overcome the pressure of the viscera pressing the rectum against the sacrum and pelvic floor, and keeping or tending to keep it flat; and thus it is that the rectum can displace visceral parts in front of it and above it.

So also, it is obvious, the connective tissue cells, situated between the pelvic walls and floor and the pelvic viscera, can only distend with fat by the pressure within these cells becoming greater than the pressure without them, the result of the pressure of adjacent cells. The variation of intra-cellular tension is a characteristic of all living cells. It occurs in virtue of their metabolism, in virtue of their being alive. It is more marked in some than in others. In muscle cells, for instance, it is phenomenal: it is in virtue of such change that muscle cells function. Since it is impossible to imagine how these connective tissue cells can increase in size, unless they do overcome the extra-cellular pressure, i.e. the pressure of other cells abutting against them—and of course the pressure of the blood within the capillaries—it is clear that, being fixed in place, they can cause the uterus and other parts to be sustained in an elevated position in the pelvis. This loading of cells with fat, increasing their volume, impossible unless surrounding cells yield before them—that is, become displaced—probably occurs during resting conditions, especially during sleep, when the pressure in the pelvis is relatively low.

The explanation of the upward growth of the uterus when pregnant is similarly easy. The uterus increases

in size because the pressure within it becomes greater than the pressure immediately outside it (between it and adjacent organs); for otherwise, a change in its volume could not occur. This pressure is caused by the actively growing fœtal cells within it. For the uterus to expand (apart from the resistance of its walls, which however immediately begin to soften, i.e. to relax, with the embedding of the embryo), it is but necessary that this pressure should be sufficient to overcome the pressure bearing upon the uterus on all sides; which, for many hours during each day—during sleep, especially in the lateral position—is low. The increase in volume of the uterus necessitates an increased space in the pelvis; the uterus must necessarily encroach upon the space occupied by the other organs, displacing them. Laterally, in front, and behind, the limit is soon reached, because of the unyielding bony walls of the pelvis and the incompressible (though deformable) viscera between. Below, between it and the pelvic floor, are other incompressible viscera; above, the incompressible viscera in the abdomen.* But the pressure in the abdomen is less than that in the pelvis—even in the supine and lateral positions—as measurements show †; the uterus therefore expands and grows upwards in the direction of least resistance.

In this, it is clear, the enlarging visceral mass must overcome the tonic contraction of the abdominal wall and diaphragm. That it should do so, is explained by the force-pump action of the pelvic floor musculature, to

* Except for the gas contained in the intestinal tract. The facts that the intestinal tract occupies such a large part of the abdomen, and that the gas within it is compressible, will, if anything, facilitate the upward growth of the uterus. Since, however, the pressure during resting conditions is not very great, this factor will not be of much influence. It may, I think, be neglected.

† The reason the pressure in the pelvis is greater than in the abdomen is a physiological one: namely, to cause the blood in the pelvic tissues to return easily to the thorax and so to the heart.

which we have referred; and by the hypertrophy of this musculature in pregnancy, whereby its compression upwards on the pelvic viscera—or its resistance to the visceral downthrust—is increased. But it is also clear, that when the abdomen is prevented from distending by the use of tightly-fitting corsets, one of two things must happen: either the increase in volume of the visceral mass, approximately incompressible, must cease; or some part of the enclosing walls must give way. Thus, the yielding and descent of the pelvic floor in such cases is easily intelligible.

Thus, it is obvious, the pressure conditions in the pelvis—the relation of the volume of the pelvic visceral mass to the capacity of the pelvic cavity, which, of course, depends on the rigidity of its walls and the resistance of its floor—are of paramount importance in the statics of the individual viscera. Whilst local variations in volume play minor rôles in the causation of variations of the pressure in the pelvis, because the enlargement of any one organ and the attendant alterations in position of the surrounding ones tend to dissipate such rises, much greater variations of pressure occur by changes in the compression of the abdominal viscera, which are transmitted to the pelvis. And these also cause the uterus to vary in position.

According to Schultze, the uterus varies in position even with the phases of respiration. On inspiration, the body of the uterus is pressed downwards on the (empty) bladder, whilst the cervix moves towards the sacrum [90]. There is clearly a gliding movement of the uterus on the bladder, the increase of pressure from the abdomen causing the uterus to pass on the bladder downwards and backwards; and probably with this, the superior wall of the bladder glides downwards and backwards over or upon the inferior wall (bladder base). And, on coughing and straining, similar but much more intense movements occur: the

increase of pressure from above is so great as to cause much more descent of the pelvic viscera and of the underlying pelvic floor.

It is obvious as a result of such impactions (e.g. cough), that a descent of the pelvic viscera collectively can only occur when the pelvic floor musculature itself descends. If the pelvic floor does not descend, the pelvic viscera collectively cannot descend; for the space is limited and the viscera possess volume and are incompressible: only could such descent occur by a displacement of the tissue fluids and blood in these viscera and in the connective tissue between them to regions of less pressure (i.e. to a higher level in the abdomen or into the thorax). And it is probable that the gentle movement caused by inspiration, although the pelvic floor may descend slightly with it, is associated with such action on the blood (and lymph) in these tissues; that as the uterus and adjacent visceral parts descend downwards and backwards, they act as a sort of force-pump, gently squeezing the blood out of the capillaries into the veins and thus assist the circulation. But besides this, it is clear, changes in the relative positions of the component parts of the pelvic visceral mass may occur—especially when the downthrust is sudden and great: that one part may descend as other parts ascend in compensation. It is easy to understand that centrally placed parts, such as the uterus, which are subjected to the full force—the axial stream—of the visceral thrust, caused, for instance, by cough, should be caused to descend downwards and backwards; whilst other parts, situated peripherally against the pelvic walls, away from the full thrust, should eddy upwards, that is ascend. In such way may be explained, as we shall see, the low position the uterus has come to occupy in the adult, and the transmutation in shape of the fœtal spherical bladder into the saucer-shaped viscus of the grown-up person.

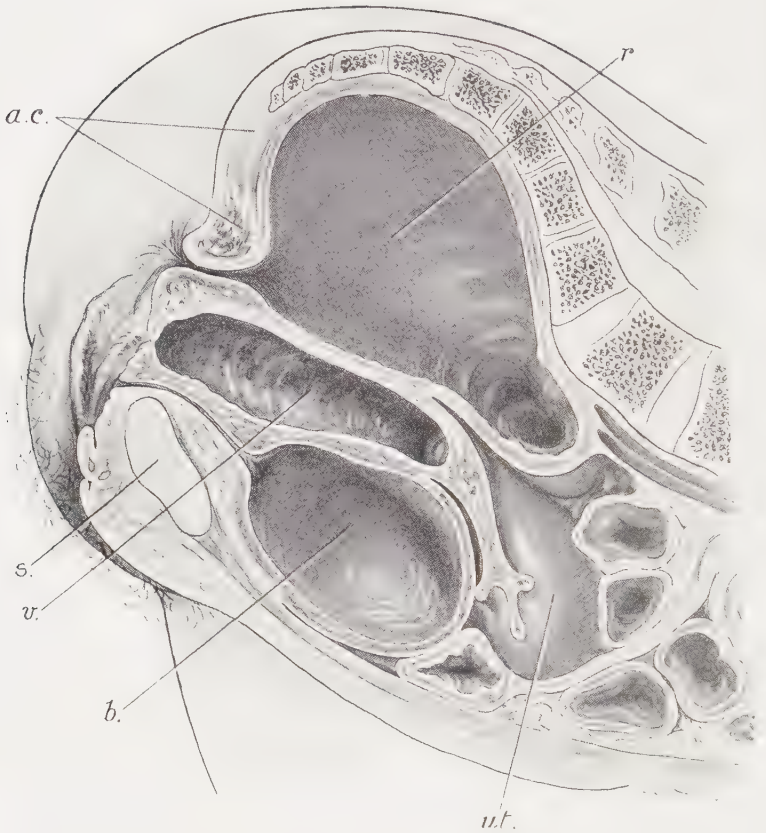

FIG. 15.—SECTION OF THE PELVIS IN THE ANTERO-POSTERIOR PLANE, THE INDIVIDUAL BEING IN THE KNEE-CHEST POSITION, SHOWING THE BALLOONING OF THE ORGANS AND THE ASSOCIATED RECESSION OF THE PELVIC FLOOR.

(After Kelly.)

Note especially the position of a.c.—the ano-coccygeal raphe. This has been depressed into the pelvis by the atmospheric pressure because the tone of the abdominal wall is much reduced in this position. The abdominal wall should perhaps be more bulged than is represented.

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So also, in the knee-chest position, the uterus becomes displaced upwards in the body towards the abdomen. But, it is clear, this cannot happen unless the pelvic floor sinks inwards, or unless some material substance, such as air, comes to occupy the space between the uterus and the pelvic floor—as may occur by its entering the vagina, rectum or bladder, causing a ballooning of these organs—for, it is certain, inversion of the individual does not cause a vacuum in the pelvis (Fig. 15). The reason the uterus gravitates towards the umbilicus in this position is, that in this position the compression of the abdominal viscera by the abdominal wall and thoracic diaphragm is much reduced or obliterated: the viscera, in virtue of their weight (in this position of the body), descend, or tend to descend, causing a corresponding bulging of the abdominal wall, just as in enteroptosis the viscera in the erect posture gravitate downwards in the abdomen because of the atonic condition of the abdominal wall in that state.

CHAPTER VI

THE POSITION OF THE UTERUS: THE INFLUENCE OF THE SO-CALLED CERVICAL LIGAMENTS

1. The Normal Displacements of the Uterus in the Adult

It is a relic of the older gynæcology that undue importance is still attached to the position of the uterus, to the neglect of the positions and indeed of the existence of the surrounding visceral parts. It is not difficult to understand this neglect. The uterus is so firm as to be easily palpable; but the other viscera are so soft as to be either impalpable bimanually, or only to be felt with the greatest difficulty. Usually, it is necessary to employ indirect means to discover their positions; for instance, it is necessary to pass the sound in order to define with any accuracy the limits of the bladder. Thus also we know that when the uterus becomes pregnant, its walls become so soft and so relaxed that it is the easiest thing in the world to overlook its existence: it is only when the contraction of its muscular wall occurs, that the presence of the organ becomes clearly perceptible. Similarly, on bimanual examination, it is difficult to realise that the non-pregnant uterus is surrounded by other visceral parts: that these, with the uterus, completely fill the pelvis: that, be they ever so soft or so displaceable, they are yet incompressible: and that, in virtue of their volume, they maintain the uterus in position. But when the contents of the pelvis are removed

in one piece and hardened and then examined, the mass they form becomes apparent. When one makes a section of such a lump and gazes at it, one becomes struck with the magnitude of the tissues surrounding the uterus. But under natural conditions within the pelvis, these tissues are none the less real in spite of not being felt distinctly; none the less incompressible in spite of being so easily displaceable.

So, too, when considering the position of the cervix, it is easy to overlook the fact that the cervix is separated from the pelvic floor by incompressible, though deformable tissues—the posterior vaginal wall, the rectum and the visceral connective tissue in which these parts are embedded;* the presence of which explains so readily why the cervix does not touch the pelvic floor. These tissues immediately come to view on section of the frozen pelvis, both in the sagittal median plane, and also transversely: inspection convinces one of their reality. But their substance is not directly perceived by palpation; because of its softness, the ease with which it can be deformed or displaced.

What we do not perceive, we do not take into account when forming opinions of other related things. So, from the neglect of this important factor—the influence of visceral parts surrounding the uterus—we see how easy a step it is to the conception that the uterus is suspended in mid-pelvis, by an aspiration from above; and, similarly, when this had been abandoned, that the reason the cervix does not touch the pelvic floor is because it is suspended by ligaments. For it is said the position of the cervix depends upon its ligaments—the thickenings of the connective tissue in which it is embedded: that if these ligaments relax, the

* With these must be included the tissue fluids and the blood in the capillaries within these tissues; which clearly are easily displaceable by pressure of the finger, etc.

cervix slides down the obliquely placed pelvic floor in virtue of its weight; and that, as a consequence, first retroversion of the uterus, and subsequently its prolapse, occurs.

Even those who object that the uterus, in spite of its weight, does not touch the pelvic floor, and yet think that it should if the pelvic floor supports it, and who for this reason believe the cervix is suspended by ligaments, will yet admit that in the adult the uterus is pretty low in the pelvis. The fundus lies on the posterior, the upper surface of the bladder: it could not get lower in this position (anteversion) without displacing the bladder. And the cervix is not far removed from that part of the pelvic floor (post-anal part) to which it is related (Fig. 4, p. 20). It is only separated from this part by the posterior vaginal wall, the rectum and some connective tissue. The cervix could not get nearer the pelvic floor than this—not even if its connective tissue had no thickenings, and was as loose as it possibly could be (as in pregnancy). The posterior vaginal wall is clearly not displaceable by the cervix: but the rectum if empty, and the connective tissue perhaps, as the result of visceral impactions, may be displaced to some extent laterally; so that during these thrusts the cervix may come nearer the pelvic floor, only however to rebound as their effect dies away.

Nor can the cervix get lower in the pelvis, that is, nearer the pubes and the pelvic floor aperture: for it can only do so by displacing the bladder. But the bladder is subjected to the same forces as is the uterus. Not only is it pressed downwards from above, and hence its shape; but gravity also attracts it, with the viscera above it, downwards: and these forces have as much effect in causing the bladder to displace the cervix backwards, as they have in tending to cause the cervix to displace the bladder forwards. Indeed, when we consider the change in position

of the pelvic viscera which occurs in the transition from the fœtal to the adult type, it becomes clear that the forces which produce this change have not only caused the flattening of the bladder from above downwards, but also are responsible for the posterior position of the cervix. Thus, it is not surprising that with the continuance of these forces, the cervix should remain in its posterior position.

And when we consider how movable is the cervix, we cannot believe the thickenings of its connective tissue can keep it in place. For though its mobility varies in extent in different cases, in many it is such as to warrant the assumption of a veritable looseness of this connective tissue—even in the non-pregnant, when thickenings are present in it—permitting movements of the cervix in all directions[105]. Above all else, this mobility shows that the thickenings of the connective tissue, which radiate outwards from the cervix towards the pelvic walls, do not cause fixation of the cervix. Clearly, the movements of the cervix which occur from before backwards and from side to side, and which bimanual examination shows are easily produced, would be impossible if the thickenings of its connective tissue were anything but slack. How, then, can these connective tissue attachments keep the cervix posterior? And the movements of which the cervix is capable in the cranio-caudal direction show these ligaments cannot suspend the organ: for these movements show the ligaments are not taut in the resting position. That such movements occur is easily demonstrated. On straining, the cervix is felt with the other viscera to descend to some extent. But, if a duck-bill speculum be inserted into the vagina, traction on the cervix by forceps can cause its displacement downwards and forwards to a much greater extent than straining in the normal can cause—indeed, the cervix can be pulled as far as the vulva.

It is true there is a limit to these movements: that the

cervix cannot be caused to descend more than a certain amount by straining in the normal; and that the cervix cannot be pulled downwards and forwards by forceps farther than the vulva. And in the latter case it is clear the limitation is caused by the connective tissue attachments; for when they are cut through further descent is possible. So that it is to be supposed that this displacement of the cervix causes a gradually increasing tension in these ligaments, which gradually comes into play, and prevents further displacement. This being admitted, the question arises whether during activity and violent movements of the body, during which the intra-abdominal pressure is greatly increased, a similar descent of the cervix, even as much as that produced by straining, occurs; and whether the reason the cervix is not extruded from the pelvis through the vagina at these times is that these connective tissue attachments come into play and retain the cervix within the pelvis, although at a lower level than in the resting condition. For it may be argued the elastic resistance of the connective tissue comes into play before such a descent is reached as traction causes.

But when the cervix is caused to descend by straining or by traction upon it, the pelvic floor is also caused to descend: in the first case, by the increased visceral thrust—the straining; in the second, by the speculum. Thus, the descent of the cervix is readily intelligible; for a space is made for it—either by it in the first case, or for it in the second—into which it is forced or is free to pass. And it is pushed or is pulled into the lower position, because its connective tissue attachments cannot prevent it being so, at least in the earlier stages of the movement. But there is this essential difference between these two cases. In the former, the increased visceral thrust causes a descent of all the pelvic viscera: not only of the cervix, but especially of the bladder, the posterior part of which, being pressed

down with a much greater force than before, with a force as great as that now acting on the cervix, must clearly tend more than ever to prevent the cervical advance—just as it does in the resting position, when the pressure from the abdomen is very much less. But in the traction experiment there is no such thrust. The other viscera are not forced down from above; the pressure to which they are exposed is low, for the experiment is done in the lateral position, or if in the position for lithotomy, during anæsthesia;* but the cervix is pulled down alone, and the traction upon it easily displaces the bladder forwards and upwards. Thus, we see the reason why the cervix does not descend more than it does during straining is not because of its connective tissue attachments: for it is still the presence of the surrounding viscera, their volume and incompressibility, and the pressure with which they collectively are thrust down upon the pelvic floor, and the resistance of this to them collectively, which determines the new position of the uterus and maintains the cervix in its new position. That the cervical connective tissue is not responsible for this, is shown by the fact that if the cervix be caught hold of by forceps, it can be pulled downwards and forwards much farther. Clearly, it is the descent of the pelvic floor caused by the straining—the visceral thrust—which allows the descent of the cervix and other visceral parts during the straining.

So also, in those postures of the body in which the intra-abdominal pressure is fairly high, as in the erect, descent of the cervix, such as occurs on straining, is impossible—because the pelvic floor remains in its usual position,†

* The low pressure in the pelvis in the lateral position and in the lithotomy during anæsthesia explains why the cervix and other visceral parts do not come down more than they do when a speculum is inserted into the vagina.

† The difference between the positions of the pelvic floor in the lateral, supine and erect postures may be neglected.

and because of the existence—the volume and incompressibility—of the surrounding viscera. The contraction of the pelvic floor musculature is increased to meet the increased pressure within the pelvis, transmitted from the abdomen, and therefore it does not descend. And this increased pressure, as before, acts not only on the cervix, but on the other pelvic viscera as well—explaining the maintenance of the cervix in its posterior position. But when sudden rises of intra-abdominal pressure occur, incidental to general activity and caused by cough, producing waves of pressure transmitted to the pelvis, changes in the relative positions of the pelvic viscera result ; for organs situated centrally are most affected. With such sudden rises, some little descent of the pelvic floor is produced ; but even apart from that, centrally situated organs—the postero-median part of the bladder and the adjacent cervix—descend with each sudden visceral thrust, and probably to an extent greater than does the pelvic floor. But they can only do so if other visceral parts, situated peripherally, ascend in compensation. But when all is said and done, the cervical descent is but little ; for the cervix is already low in the pelvis, as low indeed in the normal as it possibly can be. And the reason of the smallness of this movement is that the contraction of the pelvic floor, already sufficient to withstand the pressure in the pelvis before the sudden increase of pressure appears, is yet further increased to meet sudden pressure waves—an increased contraction occurring in spite of, and with its descent, such as this is, which the sudden visceral impact causes. But, although under such conditions some descent does occur, the pelvic floor cannot be believed to descend as much as it does on straining, when the attitude of the body is changed and adapted for this act, and the visceral thrust is sustained to produce it. Thus, too, it cannot be believed that during these times of stress, the visceral connective tissue

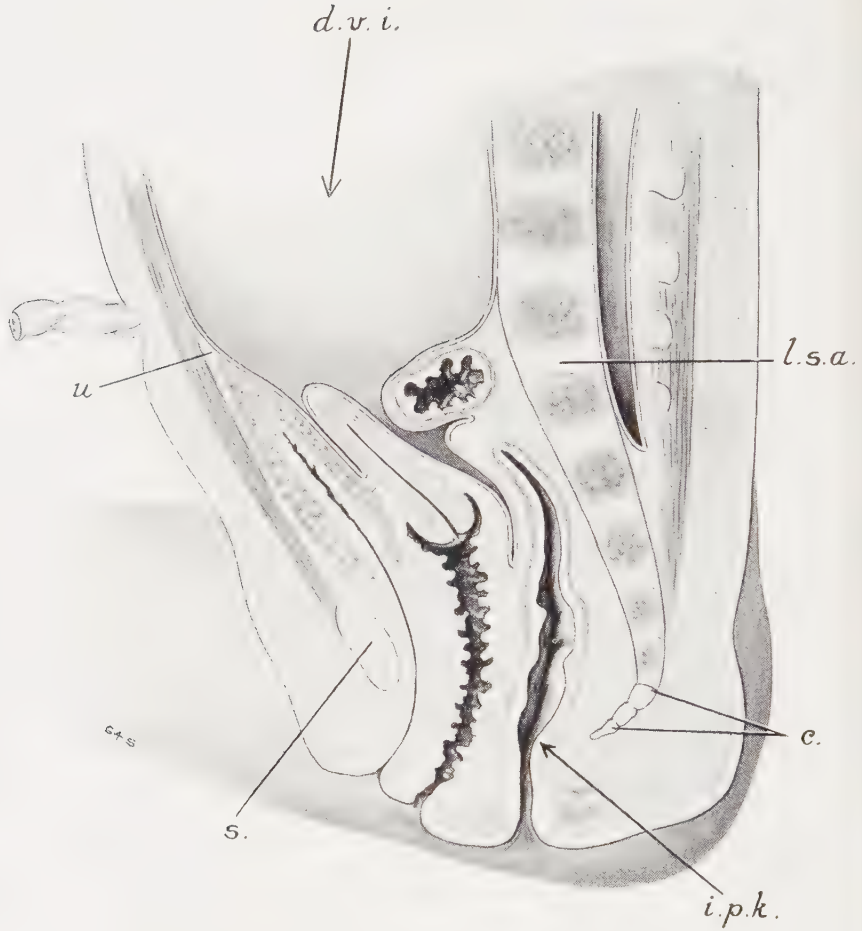

FIG. 16.—MESIAL SAGITTAL SECTION OF THE PELVIS OF THE NEW-BORN CHILD, SHOWING THE NARROW AND ELONGATED PELVIC CAVITY, OCCUPIED BY THE URETHRA, VAGINA, RECTUM AND CONNECTIVE TISSUE; THE UTERUS AND BLADDER BEING ABDOMINAL ORGANS.

Note the straightness of the sacrum, the absence of the promontory, the height of the lumbo-sacral articulation above the pubes; that the long axis of the pelvic cavity is more or less in line with that of the abdomen, and that the coccyx has already become flexed.

u. = urachus.

l.s.a. = lumbo-sacral articulation.

c. = coccyx.

i.p.k. = indication of perineal kink of rectum, which latter becomes developed into the perineal flexure—the result of the pelvis becoming bulged posteriorly and of the increasing activity of the pelvic floor.

d.v.i. = direction of visceral impaction. Their reflection from the lower abdominal wall is about to begin.

[*To face page* 93

comes into play—that it either suspends the cervix in a lower position, or prevents its extrusion from the pelvis.

2. The Ontogenetic Displacement of the Uterus

Whilst the majority of writers have asserted that the position of the cervix depends on the thickenings of its connective tissue, the reasons for which we have assigned, there is another possible interpretation of this relationship —namely, that the disposition of the thickenings of the cervical connective tissue depends upon the position of the cervix. Indeed, just as we have shown that the position of the cervix, in spite of its relative fixation in connective tissue, cannot be the determining cause of the position of the fundus, when the influence of surrounding organs in the pelvis and the pressure to which they are subjected are considered; so the view advanced by Halban and Tandler [31], that the position of the cervix is primary and the length of these thickenings secondary, is borne out by a study of their ontogeny and by the changes which occur in them in association with those more marked and more permanent changes in position of the uterus which are spoken of as displacements.

Let us consider first the natural displacement which occurs in the course of growth from the infant to the adult type, and compare the forces causing this with those acting in the adult with the maintenance of equilibrium. In the new-born child, the uterus is an abdominal organ, the cervix is high up in the pelvis and the vagina vertical—conditions readily explained by the size of the pelvis, which is relatively small (Fig. 16). The pelvic cavity is long and narrow, both from before backwards and from side to side, and is completely filled by the urethra, vagina and rectum, connective tissue, lymph and blood [6, 49]. Unlike the pelvis of the adult, it is somewhat funnel-shaped: the sacrum

is straight and vertical, the promontory not well or at all marked and is relatively much higher above the pubes than in the adult—simulating the pelvis of anthropoids. But in the adult, the uterus with the adjacent viscera have been pressed so low in the pelvis—the pelvic walls having been bulged especially posteriorly, but also I think transversely, to receive them—that the cervix, as stated, is only separated from the pelvic floor by a small mass of incompressible tissues.

Moreover, the thickenings of the connective tissue in which the cervix is embedded, so conspicuous in the adult, are not existent in the infant. They only develop slowly with the gradual growth and increasing activity of the body, appearing subsequently to the necessity for the intra-abdominal pressure, as its excursions appear and become dominant. Thus they are inconspicuous in the young, but become well marked in the adult. But it cannot be believed that because in the infant these thickenings are absent, the uterus descends in the course of growth into the pelvis in virtue of its weight. For the descent is correlated with the distension of the pelvic cavity itself: and this is not caused by the weight of the viscera, nor is it adequately explained by the causes to which the pelvic form is usually attributed. The distension of the pelvis is due, besides these, to the pressure and its excursions arising within the abdomen and transmitted to the pelvis: it is an adaptation on its own part to this pressure and its excursions. These explain the bulging posteriorly, the recession of the pelvis and the marked concavity of the sacrum, which the pull of muscles used in standing, walking and running, etc., and the weight of the trunk on the legs, do not alone explain. They also explain the enlargement of the pelvic cavity transversely, which occurs below and behind the level of the femoral thrust.

And just as the effect of the collective visceral weight cannot have sufficed to have played a part in the evolution of the adult type of pelvis, so this force is not comparable with the effect of the contractions of the abdominal wall and thoracic diaphragm in causing the positions assumed by the pelvic organs in the adult. And especially is it the great excursions of the intra-abdominal pressure—those produced more or less suddenly by correspondingly rapid variations (increase) in the contractions of these musculatures, exemplified well by cough, and occurring during activity and especially during vigorous bodily movements, so characteristic of the young—that must be held chiefly responsible. These arise high up within the abdomen, in the spacious thoracic base, where the intra-abdominal pressure is usually low and where the extensive diaphragm arches over the viscera, and are propagated downwards to the pelvis as waves of pressure. These sweep through the visceral mass, already subjected to a pressure—the pressure of posture or rest, and impinge upon these walls inferiorly. These, in so far as they yield before such pressure waves, absorb the pressure; in so far as they resist them, either reflect the waves back or deflect them in other directions, according as these waves meet the superficies at right angles or acutely.

We have already referred to the part played by the hypogastrium in the deflection of such sudden rises of pressure downwards and backwards towards the sacrum. It is these which have caused the bladder to become flattened and to extend or spread itself posteriorly in the pelvis in such a remarkable manner, just as a crumpled piece of paper is spread out by sweeping the hand over it: and to these also is to be attributed the posterior position of the cervix. For these pressure impulses are freely transmitted to the pelvis by the intestinal coils, in virtue of their great

mobility, made possible by the existence of the extensive peritoneal sac. The impact imparted to them sweeps as a wave over the anterior layer of the peritoneum, and is deflected by the lower abdominal wall backwards and downwards over the surface of the bladder. For the bladder in the infant is placed beneath this layer of the peritoneum, between it and the anterior abdominal wall, in the subperitoneal tissue, and is anchored to the umbilicus by the urachus. This, and the pressure of the other viscera behind the bladder and against it, pressing it against the lower abdominal wall, takes the place of the hand holding the crumpled paper, representing the baggy and more or less spherical infantile bladder * ; whilst the pressure wave from above, caused by the sudden increase in contraction of the thoracic diaphragm and abdominal wall, passes over it, like the other hand over the crumpled paper.

But the former forces, and the resistance of the parts already in the pelvis in virtue of their incompressibility, do not suffice to prevent the impulses from above from causing a gradual shifting of the bladder and other viscera bodily downwards. They do not suffice because the infantile pelvis is not adapted to meet such sudden visceral impactions—it is not strong enough, and since the long axis of its cavity is more or less in line with that of the abdomen, the musculature occluding its outlet is at a disadvantage. But even so, the pelvic floor muscles, even in the new-born, when considerable rises of intra-abdominal pressure occur, as on crying, react efficiently to retain the

* According to Piersol [79], in the new-born the bladder is spindle-shaped, its two poles ending respectively in the urachus and urethra ; and extends from about midway between the umbilicus and the symphysis to the level of the pelvic brim. This must be its shape when empty, but as contents collect it will become ovoid and later more or less spherical. Since pressure waves from above will have more effect on the bladder when it contains urine than when empty, I have allowed the term " spherical " in the text to stand.

pelvic viscera within the pelvis. That this is so is shown by the occurrence of those interesting cases of spina bifida, in which the lesion has involved the nerves (fourth sacral) supplying these muscles [4, 7]. In these cases, prolapse of the uterus is early caused: the fits of crying, even by the second or third day, result in the appearance and extrusion of the cervix at and through the vulva.* The considerable increase of intra-abdominal pressure, occurring repeatedly, which the fits of crying determine, is transmitted to the pelvis by means of the viscera, and finds the resistance at the pelvic outlet insufficient to withstand it, because the pelvic floor muscles normally occluding the outlet are paralysed by the lesion.

But with growth and increasing activity the intra-abdominal pressure and its excursions become greater; and the pelvis reacts to them, just as it does to the transmission of the body weight from above by the spine and to the upward resistance below by the femora and the play of all those muscles concerned when standing, walking and running begin to occur. The institution of respiration itself raises the pressure in the abdomen: for the diaphragm comes to occupy a lower position and by bulging the abdominal wall increases its tension and its tone. The volume of food taken, which gradually increases, and the increase in deposit of fat which slowly occurs, also come into play. The holding of the babe in the vertical position—dandling the infant—still further increases the pressure. To the pressure prevailing are superadded excursions caused by crying, screaming and laughing, and the lesser rises movements of the arms and legs occasion. Thus, from the beginning of extra-uterine life, viscera from the abdomen tend to enter the pelvis: and their forced entry—a gradual process—is the cause of

* In the case described by Russell Andrews, the prolapse first appeared two hours after birth [4].

the increase in the capacity of its cavity. With the learning to stand, to walk and to run, greater pressures occur. The erect posture and carriage, besides increasing the pressure, determine the development of the lumbar lordosis and sacral promontory; and by them is caused that recession of the pelvis, peculiar to man, whereby the increases in pressure can be more successfully met, its long axis being no longer in the same straight line with that of the abdomen. But the approximately hemispherical—rather, parabolic—shape of the pelvis, which is evidence of the part played by the muscular floor in its ontogeny, is due to the reaction of the pelvis itself to the pressure within and the impacts imparted to it. With growth, the pelvis bulges: but it resists the bulging, and to do so sufficiently, it increases in strength by further osseous and muscular development, by which means an equilibrium is attained. Thus it is that in the adult the bony superficies of the pelvis is able to meet successfully waves of pressure deflected against it, and its floor is capable of resisting those that impinge upon it so that those that reach the aperture do not escape.

With the change in the pelvis, a change in the positions and forms of the pelvic viscera *pari passu* occurs. The spherical infantile bladder is not only pressed downwards to a lower level; but it becomes spread out backwards over the internal aspect of the pubic arch, over the pelvic floor aperture and adjacent parts of the pelvic floor musculature, over the vagina and rectum and pelvic floor posteriorly—causing, with its own recession, a recession of these other visceral parts, and a posterior position of the cervix in the pelvis: and, acting with the other viscera above and behind it, determines the bulging of the pelvis itself, especially posteriorly but also bilaterally, and the enlargement of its cavity to which we have referred, and by which alone could such displacements of the viscera occur.

Usually, the succession of these waves, irregularly recurring, passes over the uterus; for the uterus in the infant, itself an abdominal organ, is closely applied to the bladder—it is held there by the pressure of the intestinal coils placed behind it (i.e. by the average intra-abdominal pressure at this level), and perhaps the round ligaments play a part in maintaining this position, as they do or tend to do in the adult in pregnancy. And these two forces act similarly on the uterus as they do on the bladder. Whilst the one causes the uterus to be maintained in anteversion; the other causes it to descend bodily backwards and downwards with the bladder, so that the cervix becomes posterior in the pelvis as this is enlarged. But since the bladder is the larger organ, and is constantly varying in volume, the effect on it presumably is the more important.*

In the adult, the bladder is already a pelvic organ. It is placed beneath a continuation of the peritoneum lining the anterior abdominal wall; but its upper anterior duplication does not usually extend above the symphysis pubis, except when the capacity of the pelvis is encroached on by an enlargement of other pelvic viscera. The general pressure of the viscera at this level maintains the bladder in place by pressing it against the superficies of the pelvis to which it is related; and similarly maintains the uterus applied to the upper surface of the bladder in anteversion,

* According to Disse [21] (quoted by Piersol) the descent of the young bladder is rapid during the first three years of life, slower from the fourth to the ninth year, between which and puberty little change occurs. Succeeding this period of rest, the bladder renews its descent, and by the twenty-first year has gained its definite position on the pelvic floor. Before the third year the empty bladder always remains above the symphysis; by the ninth year it has sunken below that level, but when distended the apex rises within the abdomen. In the new-born female child the uterus is situated relatively high and comes into contact with the bladder, while the vagina does not, only touching the urethra (Piersol).

causing indeed the saucer-shaped form of the latter empty organ. Upon this state of affairs, pressure waves from above, deflected by the hypogastrium downwards and backwards into the pelvis, break as a succession of irregularly recurring additional forces, sweeping over the uterus and bladder towards the sacrum, through superimposed viscera. Plainly, as long as anteversion of the uterus is assured, such waves passing over the uterus and communicated to it must tend to cause it to slide downwards and backwards over the bladder, and thus cause the cervix to become more than ever posterior in the pelvis. But this factor in the maintenance of the posterior position of the cervix can only come into play when the pelvic floor is intact and remains approximately in its normal position. If, of course, the pelvic floor descends, the bladder and cervix descend with it; and the direction of the movement from being downwards and backwards, becomes much more downwards, as on straining—the amount of visceral descent clearly depending on the degree to which the pelvic floor descends: and, since with the pelvic floor descent, the pelvic floor aperture simultaneously enlarges, especially antero-posteriorly, the cervix and the posterior limit of the aperture approach. But if the pelvic floor does not descend, or descends but slightly, and the aperture is normal, such descent and advance of the cervix, without displacement of other visceral parts, is impossible; for they are incompressible.

When we come to think of the variations of pressure which are continuously being produced within the abdomen and transmitted to the pelvis, the respiratory phases even being effective in causing variations of the pressure in the rectum and changes in the position of the uterus; that during activity the excursions are often very great, and are frequently recurring during the lifetime of the individual; and that the pelvic floor is capable of movements in the

cranio-caudal direction, and yields somewhat downwards in front of sudden visceral impactions (e.g. cough); it becomes plain that the connective tissue in which the cervix is embedded, since it is adherent to the superficies of the pelvic cavity, must be subject centrally to very frequent movements—up and down—in the direction of the visceral thrust, which must cause variations of tension, however slight, within its cells and fibrils. For the cervix, being a compact organ and centrally situated in the pelvis, is bodily and easily affected by variations of the visceral thrust from above; and thus it is easy to understand how the connective tissue in which it is embedded becomes subject to variations of tension. These frequently recurring lines of stress in this connective tissue are the cause of the appearance of the thickenings—the so-called ligaments—within it. That this is so, is shown by the fact that these thickenings begin in the region of the cervix, that they are most marked there, since this is the mobile part, and that as they pass towards the periphery, they tend to fray out and become less well marked or even disappear [34, 61]. Since the same force which causes these thickenings causes also the position of the cervix, it is plain the disposition and length of these ligaments must depend on the position of the cervix: that the position of the cervix determines the disposition of the ligaments—not that the ligaments determine the position of the cervix.

3. The Retroversion of the Uterus

And now let us consider the other displacements—the retroversion and the prolapse of the uterus. It is stated that the cervix is lower in the pelvis when the uterus is retroverted; and that this is a preliminary stage to its prolapse, and is caused by a relaxation of the cervical ligaments. When the uterus is retroverted, the cervix probably

is a little lower than when anteverted—the patient being in the erect posture—because the pelvic floor slopes a little downwards and forwards from the sacrum to the pubes. But the fact that the cervix is felt so much more readily on vaginal examination is due more to its anterior position than to its descent, a fact Schultze long ago pointed out. Whereas before, the cervix was some distance from the pelvic floor aperture, being placed above, but especially behind it; now, the cervix is much nearer the aperture, placed indeed but some little distance directly above it.

And the anterior position of the cervix in these cases is the consequence of the position of the fundus of the uterus, which is now posterior. For this displacement * is the result of the thrusting backwards and downwards of the fundus, produced by the insinuation of intestines between it and the bladder, and caused by the pressure waves acting from the abdomen to which we have already referred. In some cases, this displacement of the fundus backwards, apparently, occurs suddenly, as the result of some violent movement or fall; but retroversion is often found in nulliparous women, examined for instance because they want to know whether they are pregnant or why they are sterile, who give no such history, and these are more common than the traumatic cases. And retroversion often seems to date from a confinement, in patients in whom one can find no evidence of inflammatory infection having complicated the lying-in, to which the displacement might be attributed. In such cases, it is often said, and probably with truth in some, that the uterus was retroverted before the pregnancy, and that it simply returns to its previous position. But I see no reason why it should return merely

* Apart from those cases associated with or caused by inflammatory diseases in the pelvis, which complicate the question, and are here omitted.

because of this. But whichever variety be considered, it is plain the displacement of the fundus backwards towards the rectum, and then downwards in front of the rectum, so that the fundus is felt quite easily to be in Douglas' pouch, must have caused the simultaneous displacement forwards of the cervix, and with this a corresponding advance of the parts in front of the cervix, i.e. of the bladder and anterior vaginal wall. For the rectum is supported in place by the posterior wall of the pelvis and cannot recede, and since the uterine body possesses volume, its occupation of space posteriorly in the pelvis necessarily must have caused the dislodgment of parts previously occupying this space and their displacement elsewhere. For this reason the cervix is advanced: it is pushed downwards by the uterine body as this is pushed first backwards, and then backwards and downwards; but the downward movement of the cervix is obstructed by the pelvic floor, whereby its forward movement mainly results.

Thus the cervix is thrust forwards against the parts in front—against the bladder, itself pressed downwards by the intra-abdominal pressure. This, as we have seen, must oppose to some extent the cervical advance: its resistance is often so great as to cause a flexion of the cervix on the uterine body (retroflexion). But the bladder itself undergoes changes in form; its base becomes crumpled,* and its upper wall elevated so that it comes to occupy the space originally occupied by the body of the uterus. And the vagina, instead of passing obliquely upwards and backwards, passes much more or directly upwards; its anterior wall becoming correspondingly shortened, or thrown into horizontal folds (Winter and Ruge). Further, if the retro-

* Halban and Tandler state that such crumpling of the bladder base is artificially produced by pulling the cervix downwards and forwards with forceps, and can be seen on cystoscopic examination.

version has been of long standing, the vagina may become permanently shortened in adaptation to the anterior position of the cervix; thus not only preventing reposition of the uterus into its normal position of anteversion, but also causing the displacement to simulate a congenital condition. And other changes ultimately occur. The vesico-uterine pouch is deepened, so that it reaches as low as the anterior vaginal wall [34], showing the influence of the pressure of the intestines against it. Thus the anterior vaginal fornix comes into the same relation with the peritoneum in front of the uterus as obtains in the case of the posterior fornix under normal conditions with the peritoneum behind. All these changes become much more marked when the retroverted uterus becomes pregnant. The progressive enlargement of the uterine body forces the cervix still more forwards; and since it cannot pass downwards, it is displaced upwards, almost immediately behind the symphysis and often above it. The bladder becomes again an abdominal organ, and the urethra correspondingly elongated. The explanation of these changes is simple when we remember the viscera possess volume and are incompressible, and that the pelvic floor musculature below occludes the pelvic outlet and prevents their descent.

Whilst it is generally believed that the intra-abdominal pressure is responsible for this displacement, it is stated that relaxation of the connective tissue attachments of the cervix is a necessary condition. It is stated that if the uterus is loose as the result of such relaxation, when the bladder becomes full it pushes the uterus backwards so that its long axis coincides with the pelvic axis, and that then intestinal coils enter the utero-vesical pouch; and that when this has happened, intra-abdominal pressure completes and maintains the retroversion [28]. But it is

difficult to see how coils of intestine can enter the utero-vesical pouch merely because the bladder becomes distended. For the visceral parts about the uterus and bladder, not only those above but also those behind the uterus, between it and the posterior pelvic wall, are all compressed against each other and against the uterus and bladder in front and below; indeed, the pressure *between* all these organs is increased because of the bladder distension, as we shall see (p. 177). And this general pelvic pressure must cause the uterus to remain pressed more than ever against the bladder. But if now a wave of pressure, arising in the upper abdomen and reflected from the hypogastrium into the pelvis (full of viscera), sweeps through intestinal coils placed above and pressed down upon the distended bladder and erect uterus—and probably also through the upper parts of these organs themselves, elevated by the distension and now in the course of such pressure wave—it abuts against the fundus of the uterus, the convexity of which is not in a plane with the convexity of the distended bladder summit; and it is easy to see that such a wave may cause the fundus of the uterus to be pressed back, displacing some coils behind it upwards (peripherally), and making room in front for coils to pass downwards between it and the bladder.

It is not, as we have seen, the traction backwards of the cervix by ligaments which maintains the cervix posterior and the uterus apposed to the bladder in anteversion: but the uterus and the surrounding organs and the visceral connective tissue with all the vessels and other parts, have all become adapted to this position, as a result of the pressure within the abdomen and its variations having been transmitted to the pelvis from the moment of birth, and which in the course of growth have caused a change in its form and capacity and in the disposition of the viscera. And whether the cervical ligaments or connective tissue

be loose or not so loose, the posterior position of the cervix depends on these two factors—the pressure from above and the resistance of the pelvic floor below. For, distension of the bladder always causes the uterine body to be raised and to be pushed backwards in the pelvic cavity—it does not do this only when the uterus is loose; and, however fixed by its connective tissue attachments the cervix may be, this cannot of itself maintain the body of the uterus applied closely to the bladder, certainly not with sufficient force to prevent coils of intestine entering between. For normally the uterus, though firm, is sufficiently supple, especially in the region of the internal os, to allow a flexion of the body on the cervix—if not suddenly, at least gradually. The body of the uterus can only be maintained applied to the bladder by pressure of intestinal coils behind the uterus. And retroversion can only occur when these are forced upwards. This, as we have seen, may occur peripherally—at the sides of the pelvic cavity away from the axial stream of pressure, the pressure wave. But the extent of this upward movement of peripheral visceral parts must be much less in individuals in whom the general intra-abdominal pressure—the pressure prevailing before the advent of the pressure-wave—is good, than in those in whom it is not so good. And as a matter of fact, we find retroversion is much more often present, and can often be caused bimanually *easily*,* in individuals with lax body-wall musculatures—those in whom, for instance, coughing causes marked bulging of the lower abdominal wall laterally, outside the recti—than in women with well-developed musculatures. Besides, the suggestion that the fixation of the cervix by its attachments prevents retroversion is

* I have often surprised myself by the ease with which a uterus in the normal position of anteversion can be caused to become retroverted by bimanual manipulation in such cases, the patient lying on her back at ease, without causing the slightest pain.

insupportable when we recognise that distension of the bladder causes the cervix itself to become more posterior and higher in the pelvis ; for this robs the connective tissue of its supposed effect, and is moreover a change of position which does not favour retroversion, but which opposes it.

But in the case of retroversion produced by accident, the resulting pain or ache, which is said to occur, may be adduced as proof that some traction has been put on these connective tissue attachments ; and perhaps the pain may be due to such traction. And it may be argued from this that the connective tissue does tend to maintain the cervix posterior. Often, too, the displacement appears to occur gradually, so that on examination, even when the bladder is not distended, the uterus is found neither anteverted nor retroverted, but in a position more or less vertical (in relation to the erect posture) * ; and these cases may be cited as showing that the cervix is held posterior by its ligaments. It may be admitted at once that the connective tissue attachments do tend to anchor the parts, do limit the movements. They prevent, as we have seen, the traction of the cervix by forceps further than the vulva : that is, they allow it as far as the pubic arch ! If this be the normal condition, we cannot believe the " tonicity " or elasticity of the connective tissue attachments of the cervix prevents retroversion, or that retroversion is a consequence of their relaxation, much less is caused by it. For were this so, the mobility of the normal cervix, which depends on this very condition, is such that every normal woman should have a retroverted uterus, and that this should be the normal uterine position.

When we recognise that the pelvis is full of viscera and visceral parts, that all possess volume and are incompressible, and that all besides being pressed against each other

* Some authors call this " retroversion," naming the displacement of the fundus in Douglas' pouch " retroflexion."

are also adherent (except where the invaginations of the peritoneal sac intervene) to one another and to the enclosing walls and floor of the pelvic cavity, it is not surprising that all the visceral parts surrounding the uterus should tend to resist a displacement of the uterus so great as its complete retroversion, especially if suddenly induced and more especially when the general intra-abdominal pressure is good—i.e. in well-developed women with good abdominal walls; that not only the *thickenings* of this visceral connective tissue should resist it, but the other parts of the connective tissue and the organs themselves. For such a change produces marked changes in the positions, and so in the forms of the surrounding organs, connective tissue and peritoneal folds; and since the uterus is much firmer than these, it must cause extensive changes in the tension of surrounding parts and an interference of the blood-flow through them. Thus it is not surprising some discomfort should occur when an anteverted uterus suddenly becomes retroverted. But these cases are uncommon. Indeed, their occurrence is looked upon with suspicion [96]. And the reason is plain; for in well-developed women, the intra-abdominal pressure is so high as to prevent in great measure this displacement. Even when such occurs, it is by no means certain that the alleged ache or pain is due to traction of the cervical ligaments. It is generally imputed to a congestion of the uterus, caused by the compression of its veins by the utero-sacral folds. But this cannot happen unless the tension of these folds be increased; and the resulting congestion presumably would take some little time to arise. The pain may be due to the tension itself of these folds. Such tension would tend to prevent further advance of the cervix; but in the normal position of the uterus these folds are not tense, and they are not thought to be effective in maintaining the cervix posterior.

And much depends on the size and rigidity of the uterus. The uterus of virgins and of nulliparous women, in whom retroversion is so often found, is so small and so mobile that retroversion could easily be produced without causing any traction on the connective tissue attachments of the uterus—and surely this is the normal condition. But in the larger uterus of multiparæ, in whom the rigidity of the uterus too is often much greater, a sudden thrusting of the fundus downwards into Douglas' pouch is much more likely to cause traction. But it is also much more likely to cause impaction—contact pressure changes—equally effective in causing pain. But even if so, even if in some cases the connective tissue attachments of the cervix are so disposed as to offer a resistance to the advance of the cervix, the occurrence of the displacement shows they have been unable to prevent the displacement. For the displacement is caused by a force acting on the fundus and therefore having a considerable leverage on the cervix. And in time, if the displacement continues, the pain or ache, if due to such traction, passes away, as the connective tissue attachments adapt themselves to the new conditions. The continuous tension causes their elongation or relaxation. Clearly, this is a secondary change: it is not primary, it is not the cause or a condition of the retroversion.

And even if such obstruction to the cervical advance is a cause of the gradual occurrence of retroversion, there is at least another explanation of the vertical position of the uterus. For though the presence of intestinal coils in the utero-vesical pouch, and the pressure waves from the hypogastrium to which they are subjected, press the uterus backwards; other parts of the intestinal tract are still behind the uterus, and these as long as they are subjected to the same pressure waves, tend to keep the fundus anterior, and thus the vertical position persists. But, besides this, the same forces as before act on the bladder, pressing it

downwards, tending to keep it in place and flattened out; and the posterior part of this, pressing against the cervix, must tend to prevent the cervical advance. But usually, the sudden rises of pressure, reflected from the hypogastrium, passing over the bladder against the vertically standing uterus at length prevail—the uterus clearly is at a disadvantage—so it is not surprising that retroversion should ultimately occur, provided the uterus be small enough. With each step in this change, an adaptation of all the surrounding parts occurs, not only of the visceral connective tissue, but of the organs as well.

Nor when retroversion follows labour in a woman in whom the uterus was anteverted previously to the pregnancy, can the cervical connective tissue have any effect in causing the retroversion; because of its laxity in pregnant women. The condition of the connective tissue, as in the other cases, permits the retroversion, but does not cause it. It is probable during the involution of the parts after parturition, that the uterus retains the vertical position* as it subsides into the pelvis, intestinal coils getting in front of it. This may be explained by the continued dorsal posture of the patient, and the laxity of the parturient abdominal wall, which may permit the difference in specific gravity between the involuting uterus and the gas-contained intestines to come into play—a condition which does not obtain when the intra-abdominal pressure is good. When such happens, with the return to active life, the pressure from above acting on the anterior surface of the uterus readily causes its retroversion.

In the majority of cases (free from inflammatory disease) in which retroversion is discovered, it causes no symptoms, for the traumatic cases are rare. The condition is discovered accidentally. Nor if the uterus is replaced in

* *i.e.* with its long axis parallel with the long axis of the body.

anteversion, is it easy to keep it there. For all the parts have become adapted to its new position, and if the retroversion be of long standing, they tend to return to these positions when displaced from them. In the traumatic cases, if the cervical ligaments have been stretched, they relax and elongate; in the other cases they gradually adapt themselves to the position of the uterus; in the lying-in cases, the reappearance of the cervical ligaments, which the return to active life causes, has been such that the *status quo* remains. The ligaments take up a position and a length depending on the position of the uterus.

4. Prolapse

It is of course true that the cervix must become anterior before it can become prolapsed—that is, before it can be extruded through the vaginal orifice. But it is an error to assume that in ordinary cases its anterior position in cases of retroversion facilitates or is in any way a cause of prolapse. This assumption is belied by clinical experience. The frequency of uncomplicated cases of retroversion* without any prolapse—that is, without any protrusion and without any of the bearing-down pains associated with the production of prolapse—cases which when watched for years never give rise to prolapse, shows that retroversion *per se* is not a cause of prolapse. It is just as reasonable (or as unreasonable) to suppose that the anterior position of the bladder, placed as it is immediately above the pelvic floor aperture—that is, just over it—is a cause of cystocele.

Prolapse † is produced by two correlated causes: (1) by

* That is, free from inflammatory disease.

† By prolapse is to be understood an extrusion of parts of the pelvic viscera, irrespective of the organ or organs involved, through the pelvic floor aperture—the disease is one disease, not several

an increased pressure in the pelvis (as occurs in the newborn on crying); and (2) by an insufficient resistance of the pelvic floor musculature to this pressure (as when the musculature is paralysed as in spina bifida). These respectively, are the exciting and predisposing causes of prolapse. In the majority of cases (i.e. in the adult), the exciting cause is the frequent repetition of great or of considerable rises of pressure, often produced suddenly, some often sustained, the concomitant of arduous manual labour, of the straining incidental to constipation, or of the incessant cough of chronic respiratory diseases. But occasionally, the exciting cause is a rise of the *average* pressure in the pelvis—apart, that is, from increments of that pressure, the result of activity, etc., which are superadded and which may or may not be unduly high. Such a rise is found in robust corpulent people, and is due to the increase of the general pressure in the abdomen [75], which necessarily affects the pressure in the pelvis. The occurrence of corpulency is occasionally associated with the appearance of prolapse, when the predisposing cause coexists, and dieting of these patients may materially assist in their cure. The advance of pregnancy, like that of corpulency, produces an increase of pressure in the abdomen and pelvis; and pregnancy, like corpulency, is sometimes associated with prolapse of the cervix [106] or with cystocele [84]. * In the same way, the wearing of an

diseases. Usually, the extrusion occurs through the vaginal orifice; and includes cystocele and rectocele as well as prolapse of the uterus. But it may occur through the urethra and its orifice, or through the anal canal and anus.

* The same factor, due to accumulation of intestinal contents, has been assigned as a cause of the prolapse in a case of imperforate rectum in which prolapse occurred forty-eight hours after birth [3]. In this case " there was increased intra-abdominal pressure, but no spina bifida. When this pressure due to imperforate rectum, had been relieved by colotomy, the uterus remained in its normal position " (Russell Andrews [1]).

abdominal belt, by causing an increased pressure in the abdomen and so in the pelvis, may usher in all the symptoms of impending prolapse (bearing-down pains, etc.), which disappear when the belt is left off. More rarely, the increased pelvic pressure arises apart from abdominal conditions; and is then determined by the progressive increase in volume of a tumour, such as a growing ovarian cyst, which caught beneath the promontory is unable to enter the abdomen.

The paralysis of the levator ani muscles is a rare predisposing cause of prolapse. Commonly, their inefficiency results from an injury sustained during childbirth; or it results from a weakness, due to malnutrition. The injury of childbirth affects especially that part of the pelvic floor musculature forming the sphincteric mechanism about the pelvic floor aperture (pubo-rectalis) through which the child passes. It produces a permanent enlargement of that aperture, especially transversely [2]; and a less efficient closure of the aperture by a less adequate compression of its obturator against the pubes is the natural result. Commonly—and happily—during the majority of births, the injury of the pubo-rectalis is insufficient to allow subsequently the extrusion of visceral parts. Perhaps in some cases, the muscle escapes injury: in others, the injury is insignificant, or during the lying-in and subsequent easy time, repair takes place, so that with the resumption of active life, the pubo-rectalis again functions efficiently. Even should prolapse occur, it is plain the part of the floor behind the aperture, remaining efficient, may be able to maintain its normal position; its descent, provided the pressure conditions in the pelvis are normal, can only occur when it also has suffered whether by a laceration or a prolonged and excessive stretching of its fibres [89]. Childbirth, however, is much less likely to affect the posterior parts of the pelvic floor in either of

these ways than the anterior parts which from their position and anterior attachments are much more liable to injury. But when prolapse persists and advances, the posterior parts of the pelvic floor are secondarily affected, so that in old standing cases they are found atrophic.

Unlike the injury consequent on childbirth, in so-called atonia, the whole musculature of the pelvic floor, from the start of the disease, is involved—the superficies supporting the pelvic visceral mass (coccygei and ilio-coccygei) being as much affected as the parts in front guarding the aperture (pubo-coccygei and pubo-rectalis). Not only does a descent of the whole pelvic floor occur, but often the aperture is enlarged as well—but in this case, the enlargement occurs especially antero-posteriorly. In such cases, it is clear, the rises of visceral pressure in the pelvis due to manual labour, straining and cough, are much more likely to cause visceral extrusion than when the musculature is normal. But sometimes no prolapse is found, even when the descent of the floor is considerable. Two explanations may be given for this. On the one hand, the rises of pressure due to activity, etc., are often less than occur in the normal; for in atonia, the other body wall muscles (diaphragm and abdominal wall) are also involved, and the individuals are weaker and less capable of activity. On the other hand, the superficies of the floor is more affected posteriorly by impacts from above than the part in front; and the more inefficient it is, the more it yields to such impacts, the more are the pressure waves absorbed and in less degree reflected or propagated to the aperture. Much less likely is the pubo-rectalis (situated beneath the pubo-coccygei anteriorly) to suffer from such pressure changes, since it is unattached to the coccyx behind or to the ano-coccygeal raphe. Thus, it is not surprising it should be able to continue to function more or less adequately—that is, sufficiently to meet the needs

of its particular case. The pubo-rectalis does become impaired, just as do all the other muscles of the body; but only when visceral parts bulge into the aperture is it greatly affected by the pressure conditions within the pelvis. Clearly, if the muscular boundaries of the aperture (pubo-coccygei as well as pubo-rectalis) have previously been injured in some childbirth, yet insufficiently to allow visceral extrusion (a common complication), the advent of atonia would be much more likely to result in prolapse.

The exciting and predisposing causes of prolapse, clearly, are correlated. If an extensive injury of the pelvic floor has occurred, a lesser force, or a succession of lesser forces from above will occasion prolapse than will be able to cause it when the injury of childbirth is less severe. Thus, whilst hard manual labour will in one case be insufficient to produce prolapse, even ordinary exercise in another may induce it. And, if manual labour causes its symptoms, a less arduous life often relieves them. Similarly, it is easy to understand how a woman during the child-bearing period may remain free from prolapse, which only supervenes when she grows fat. So also we see that the occurrence of prolapse with tumour formation is intelligible even when no predisposing lesion can be discovered to have affected the pelvic floor musculature before the tumour began. These cases demonstrate plainly enough that prolapse is caused by an increase of pressure in the pelvis. Strange, then, is it to find that retroversion of the uterus is regarded as preliminary to and related in a causal way with its prolapse; and . . . that "increased intra-abdominal pressure . . . does not loosen normally fixed organs and thus is not a cause of prolapse. . . ." (Fothergill [28]).

When prolapse of the bladder (cystocele) occurs, it occurs because the bladder is already there just above the aperture

when the causes which produce prolapse ensue—e.g. excessive manual labour *plus* an enlarged pelvic floor aperture. Under such conditions, the bladder is thrust down into the aperture, invaginating the anterior vaginal wall in front of it—it first dilates the aperture, and is then partially extruded through it. Whichever organ is first thrust into the aperture, it at first prevents the entrance of other organs; for it blocks the aperture, enlarged by the extrusion (as in atonia) or by the injury. The sphincter muscle about the aperture—at the sides and behind—although weakened or injured, still resists dilatation and tends to prevent further visceral extrusion by grasping the obturator now augmented by the prolapsed part—just as in the same way in the normal, it grasps the visceral canals between it and the pubic arch, preventing extrusion. But when the cervix is anterior, either because the uterus is already retroverted, or because the pouch of Douglas has become dilated by intestines, and the causes which produce prolapse appear, the cervix is first extruded, because it is over the aperture. The anterior position of the cervix is only possible by displacement of the bladder. The bladder has already been pushed to the front by the causes which produce the anterior position of the cervix, it has been displaced forwards and upwards out of the way, and the cervix is pressed down into the aperture, because it is there over the aperture. But the anterior position of the cervix is not a cause of its prolapse any more than the normal position of the bladder is a cause of cystocele: for the cervix can be anterior for years, even during active life, without prolapse being induced.

Or, if the posterior vaginal wall, invaginated by the intestinal coils within a dilated Douglas' pouch, is first extruded, it is plain it must first have displaced upwards the cervix and the bladder as well. The occurrence of these cases is explained by the frequent repetition of suddenly

occurring rises of intra-abdominal pressure, such as that produced by cough, and affords another example of the effect of visceral impactions in causing displacements of visceral parts situated peripherally to the axial stream of pressure. Clearly, in the initial stages, Douglas's pouch can only be dilated by displacing the normally apposed surfaces —the rectum is pressed backwards as much as possible and flattened; but the principal effect is in front, where the uterus and bladder are displaced forwards. With the advance of the condition, the distending posterior vaginal wall is directed anteriorly by the pelvic floor; and is insinuated between the rectum supported by the resisting musculature below, and the cervix and the anterior vaginal wall with the bladder above and in front. If it is to enter the aperture, up till now not dilated, it can only do so by forcing these other parts upwards and out of the way. If these parts are pressed downwards with sufficient force, they prevent the advance of the posterior vaginal wall: if they first are extruded, their extrusion at first prevents the extrusion of the posterior vaginal wall. Clearly, if the pelvic floor aperture has already been enlarged and the muscle guarding it is less efficient than normal, prolapse of the cervix with or without cystocele is much more likely to result; for they also are depressed by the impact of cough, though to a less degree than the intestinal coils, and in the early stages are immediately over the aperture. But if the pelvic floor be intact, the cervix and bladder cannot escape, and are displaced upwards by the advancing posterior vaginal wall, which distended by intestines is bulged forwards by the same impact. Ultimately, this may reach the aperture and overcome the muscle about it, just as happens in the case of any other hernia. Since the former condition of the pelvic floor is much the commoner, prolapse of the cervix and bladder base is much more common than vaginal

enterocele, even though incessant cough be the exciting cause of this condition [40].

It is the same with rectocele. Here the anterior rectal wall becomes dilated by a recurring increase of pressure within it, the result of the habit of straining at stool in the absence of the natural desire to defæcate or when the natural stimulus from the bowel has passed away—that is, in the absence of peristalsis, when the musculature of the gut wall is uncontracted or is insufficiently contracted. In these cases, the rectum is distended with contents, and the space it occupies in the pelvis is often considerable. The effect of the sustained increased pressure from above (i.e. of the straining) is to press the rectal contents forwards; and since the gut musculature is inactive whilst the anal sphincters remain contracted, the fæces instead of escaping per anum bulge the anterior rectal wall in the region of the perineal flexure and upper part of the anal canal forwards, towards the aperture dilated by the straining and unoccupied by other parts. Clearly, this bulging forwards of the rectum must of necessity displace the visceral parts in front of it—the posterior vaginal wall, the anterior vaginal wall with the bladder and uterus above it—if it is to enter the aperture, dilated by the straining, and to become extruded. But these also are pressed downwards by the straining and oppose the advance of the rectum. Thus it is that when the aperture is larger than normal, habitual straining at stool is more likely to induce cystocele and extrusion of the cervix than rectocele which is for this reason a relatively infrequent variety of prolapse.

There is but one condition in which retroversion of the uterus seems to facilitate prolapse of the organ, and that is in those uncommon cases associated with an impacted and growing tumour in the pelvis. If an ovarian cyst develops above the retroverted uterus, and gets caught beneath the promontory so that it cannot enter the abdo-

men, prolapse of the cervix is likely to occur, even though the pelvic floor was previously normal and its aperture had not been injured by childbirth ; but if it develop above and behind an anteverted uterus, no prolapse may result. But, clearly, in these cases, it cannot be maintained that the retroversion of the uterus is a cause of its extrusion. The cause of the prolapse is the growth of the tumour, which occasions a very great increase of pressure in the pelvis. Not only is the pressure within the cyst itself high ; but the pressure outside it, between it and surrounding organs, has become greatly increased because of the growth of the cyst and its inability to enter the abdomen. We have evidence of this pressure in the considerable descent of the pelvic floor which occurs. Moreover, the removal of the ovarian cyst, by removing the increased pressure in the pelvis, cures the prolapse—of which after the operation no return occurs. But the reason the cervix is extruded is because it is already over or in close proximity to the pelvic floor aperture before the causes which produced its prolapse arose ; and the gradually increasing pressure in the pelvis, which is sufficient to bulge the whole pelvic floor downwards, has little difficulty in insinuating the cervix into the pelvic floor aperture, dilating it in a wedge-like sort of way. But marked prolapse in these cases does not occur : usually the cervix only is extruded. On the other hand, when an ovarian cyst grows above the anteverted uterus and similarly becomes impacted, it presses the anteverted uterus on to the bladder, and this viscus is flattened over the pelvic floor aperture. Escape of the uterus, clearly, is impossible, but some pouching of the bladder may occur. But if the pelvic floor aperture is otherwise normal, and has not been injured by childbirth, that is, has not become enlarged transversely, no prolapse in this case need happen. The bladder has become spread out by the increased pelvic

pressure, just as it does in pregnancy: it has crept up the lateral walls of the pelvis and also anteriorly into the abdomen, and the part related to the aperture is stretched over it. There is no wedge here to be insinuated into the aperture, which though enlarged antero-posteriorly is but little increased transversely; and whilst the posterior part of the floor may have given way considerably, the pubo-rectalis being less influenced by the pelvic pressure than other parts of the musculature, because it is beneath the pubo-coccygei and is unattached to the coccyx or ano-coccygeal raphe, is obviously capable of functioning and of preventing visceral escape.

Just as it is obvious that the cervix cannot remain anterior when the uterus is retroverted without secondary changes in its connective tissue attachments occurring in adaptation to the new position of the uterus, so it is obvious extrusion of the cervix from the pelvis cannot occur without similar changes being produced in these attachments. Their elongation occurs. With prolapse of the cervix, Mackenrodt's ligaments necessarily become longer: when prolapse of the uterus is complete, the utero-sacral ligaments also are greatly lengthened (Fig. 17). The same applies to the pubo-vesical ligaments. When the bladder base is so prolapsed that the proximal part of the urethra, instead of running upwards and inwards into the pelvis, passes downwards and backwards into the cystocele, these ligaments are similarly affected.

Because of such changes, it is concluded that prolapse is due to a yielding of these ligaments. Such yielding is said to be the result, almost invariably, of pregnancy and parturition (Fothergill) [28]. But this is an unsatisfactory and obviously untenable explanation. For prolapse does not always immediately follow the return to active life after labour: indeed, it is in the minority of cases that

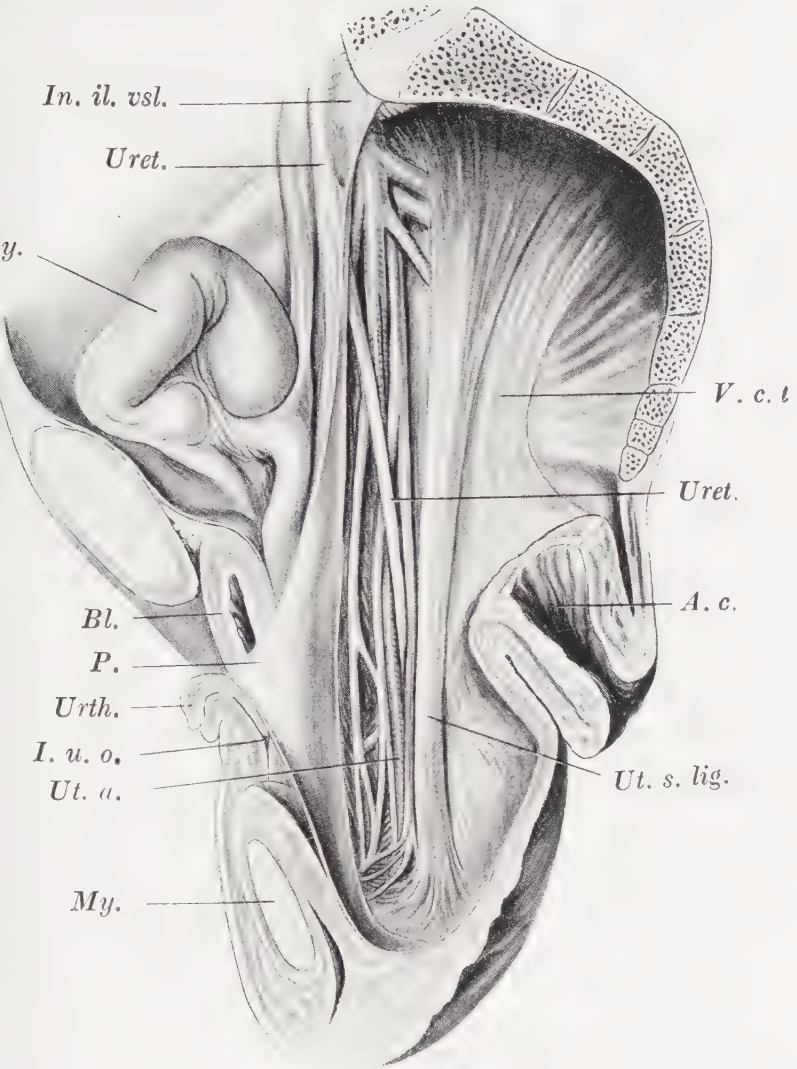

FIG. 17.—A VIEW OF THE RIGHT HALF OF A PELVIS DISSECTED TO SHOW THE UTERINE VESSELS, URETER AND UTERO-SACRAL LIGAMENT FROM A CASE OF ADVANCED PROLAPSE—THE SAME AS THAT FROM WHICH FIG. 3 (P. 18) WAS OBTAINED.

(From Halban and Tandler.)

The uterine vessels, ureter and utero-sacral ligament are all greatly elongated. The utero-sacral ligament is hypertrophied. The pubo-vesical ligament is not shown; but it is seen in Fig. 3 and is also elongated and thickened.

The ureter, in its proximal (intra-pelvic) part, is not dilated (as it often is in partial prolapse): and there is no evidence of constriction of the prolapsed mass in the region of its neck by the pelvic floor musculature, as there is in cases of partial prolapse (Fig. 20, p. 242). In this case, in which the prolapse is complete and the pelvic floor atrophic, the prolapsed mass is suspended by all the structures which attach it to the pelvis and pelvic contents.

In. il. vsl. = Int. iliac vessels.
Uret. = Ureter.
Hy. = Hydrosalpinx.
Bl. = Bladder.
P. = Peritoneum.
Urth. = Urethra.
I. u. o. = Int. urethral orifice.
Ut. a. = Uterine artery.
My. = Myoma.
Ut. s. lig. = Utero-sacral lig.
A. c. = Anal canal.
V. c. t. = Visceral connective tissue.

To face page 120

this happens. It is not at all uncommon in women who have never borne children; and in multiparæ, it frequently first appears long after the last parturition—which therefore come into line with the first class of case. This looseness and relaxation of the connective tissue occurs in all cases of pregnancy: but subsequently the thickenings of the connective tissue reappear. They have to be cut through for the removal of the uterus in multiparæ as in nulliparæ. Their absence or their presence neither explains the prolapse nor the maintenance of the uterus in its normal position. Nor does such a conception explain the different varieties of prolapse.

But the thickening of these so-called ligaments, sometimes found in prolapse, may be put forward in support of the idea that under normal conditions they support the uterus—it may be supposed that the uterus has prolapsed because of some intercurrent disease affecting the ligaments, and that the ligaments subsequently have hypertrophied and so stayed the displacement. In Fig. 17, the utero-sacral ligament is displayed as greatly developed—it is not only elongated; it appears hypertrophied. But in the case from which this drawing was obtained, the pelvic floor was highly atrophic (Fig. 3, p. 18): it is plain, it could play little part in sustaining the prolapse; and the thickening of the utero-sacral ligaments is intelligible. But in less advanced cases, in which the pelvic floor is much better developed—in which, therefore, it has been able to prevent the advance of the disease (as in Fig. 25, p. 351)—such a condition does not obtain.* According to Halban and Tandler, in cases of partial prolapse, the utero-sacral ligaments are either unaltered, or else are lengthened (often considerably) and so thin as to be unrecognisable.†

* See footnote p. 240.

† This is especially so in cases of Douglas' hernia with elongation of the anteverted uterus, in which clearly a stretching of the utero-sacral ligaments must occur. (Halban and Tandler [34].)

In the same way is to be explained the thickening of the pubo-vesical ligaments, and of other parts of the visceral connective tissue when this occurs.

The reason these ligaments do not elongate in the normal is that they are prevented from doing so by the pelvic floor placed below which resists the visceral down-thrust. The pelvic floor prevents the descent of the viscera, and traction on the ligaments. Obviously, if the musculature becomes inefficient, it can no longer play this part; and this is the reason that prolapse, and with it an elongation of these ligaments, occurs. Similarly, when prolapse has occurred, if the pelvic floor is unable to prevent the prolapse increasing, the connective tissue attachments of the prolapsed visceral parts obviously come into play—thickening: and ultimately by their means the prolapse is suspended.

That, as a matter of fact, the pelvic floor musculature has become inefficient in prolapse is shown by its dissection in these cases (Halban and Tandler [34]). Either the aperture is enlarged by injury, or else the whole musculature atrophied and sunken. In the former case, when the aperture is enlarged, it is clear, the muscle that remains may still be well developed; just as we get inguinal hernia in athletes—that is to say, a local weakness in the muscular parietes of the abdomen when these are otherwise well developed. But the similar combination in prolapse has caused some confusion. In these cases, however, one finds clinically that the pelvic floor aperture is larger than normal, as is shown by its measurement with callipers, or in other ways, as Adolph has demonstrated [2].

When the injury to the pelvic floor has happened (as from childbirth) and the supposed value of the cervical ligaments as suspensory agents is put to the test, they are found to fail. No woman who has a considerably enlarged pelvic floor aperture and who leads an active

life, and is yet free from prolapse, has been exhibited.* Visceral extrusion is inevitably and universally the result. On the other hand, plenty of patients are seen in whom the cervical connective tissue attachments are so loose that the uterus can be moved on bimanual examination in all directions, and who are leading active lives and yet in them there is no evidence of prolapse and none of its symptoms. The reader scarcely requires to be reminded that such mobility of the uterus is the normal condition.

Thus also we see that in prolapse the "ligaments" adapt themselves to the position of the uterus.

* It is presupposed that no pessary or other support is worn.

CHAPTER VII

THE MEDIUM IN WHICH THE UTERUS IS POISED

1. General Considerations: The Conditions of the Mobility of the Uterus: The Deformability of the Medium

When, instead of considering the statics of individual viscera, as though organs were kept in place irrespective of the existence of neighbouring organs and of the influence they must physically exert, we come to regard the contents of the pelvic cavity collectively, as a heterogeneous mass of tissues, squeezed into this cavity from the abdomen, the forces at play in the maintenance of the individual organs in their several positions become easier to understand. By such a mental readjustment, which the inspection of sections through the frozen body immediately justifies, and measurements of the intra-pelvic pressure in the living confirm, we begin to perceive how the position of any one organ, in virtue of its volume, must affect the positions of other adjacent organs; and, conversely, how its own position must depend and be adapted to the positions and volumes of surrounding parts. And, moreover, it becomes clear that the effect of the weight of any one organ—such as it may measure in air—must be annulled more or less as a factor in the causation and maintenance of its position, because of the effect of the weights of the other viscera surrounding, abutting and pressing upon it. Clearly, this must be so in the case of different viscera of the same specific gravity: in the case of those of which

the specific gravity is different, the effect of this difference will be less, the greater the pressure by which all are compressed; and will only come into evidence when this is considerably reduced.

Turning to the uterus with such a conception, the first thing that strikes one is that the organ is embedded in the midst of the pelvic pole of the visceral mass; and that the collection of tissues about it, between it and the pelvic walls and pelvic floor, may be regarded as a medium in which it is poised. When looked at in this way, the influence of surrounding organs—the medium—on the uterus, and the way in which they affect its position—maintaining it or changing it—becomes apparent; whilst the necessity for a suspension of the uterus by ligaments simultaneously disappears.

That such a conception is tenable is fast becoming evident. The pelvis, we have seen, does form a container; adequate in every respect, not only to contain the viscera within it, but to withstand the pressure and its variations to which the viscera are exposed, and to retain them in spite of it and its changes. The pelvis has become adapted in its ontogenetic evolution to this pressure and its excursions; and in adult life it undergoes further adaptive changes—for instance, in pregnancy—whereby an equilibrium is maintained. Instead of a funnel, it is basin-like in form; capacious and strong. Its floor, in virtue of its striated muscle, is capable of resisting the pressure; and, under certain circumstances, of being overcome by it. Thus, whilst it presents a pressure-bearing superficies to the viscera collectively, it at the same time permits the canals to function which pass through its aperture. This, as is so commonly believed, is not a patent foramen, but is potential only; for under ordinary circumstances, and in the normal, it is occluded by the visceral canals—in virtue of their bulk, where and as they pass through it, and in

virtue of their compression at this place against the pubic arch in front by the muscle forming the lateral and posterior boundaries of the aperture. In spite of its position, in the most dependent part of the pelvis, and so apparently most dangerously placed, it is, as we have seen, on the contrary, in the most advantageous situation. For it is placed as far forwards as possible in the floor, far out of reach of the impactions from the abdomen. It is these which have caused the great changes in the positions and forms of the pelvic viscera in the course of ontogeny: and it is these which, in prolapse, are usually responsible for the visceral extrusion. In the ordinary extended position of the spine, they are reflected or directed downwards and backwards into the pelvis, and impinge on the internal superficies of the pelvis posteriorly, considerably behind the pelvic floor aperture. This they only reach by further reflection or diffusion, by which they are weakened. Thus, the aperture, in virtue of its anterior position, is preserved from the direct effect of impactions; and the viscera abutting upon it are less liable to extrusion.

And, in considering the position of the uterus, we have seen this clearly depends, at any one moment of time, on the existence of the surrounding visceral parts—in virtue of their volume and incompressibility; on the support given to all by the superficies of the pelvis—in virtue of its resistance; and on the variations of pressure to which all are exposed—not on the thickenings of the connective tissue in which the uterus is embedded. So also, we have seen that change in the position of the uterus depends on changes in these visceral parts surrounding, abutting and pressing upon it. Clearly, to maintain our conception and prove our parallel, the visceral parts about the uterus—the medium—must be easily deformable, or displaceable to other parts of the cavity—the container—in which all are placed, for otherwise changes in the position of the

uterus could not readily occur: and, further, changes of pressure in one part of the medium must influence the pressure in other parts. If this be so, it is clear the medium in the body is comparable in some measure with the medium used in our experiment, which we ventured earlier in this book to assume: from whence it will follow that the physical nature of the medium in the pelvis affords the explanation of how it is the uterus is supported by the pelvic floor, in spite of not touching, not resting upon it; and how it is the uterus is so readily mobile or displaceable. It remains, then, to consider the medium in which the uterus is poised: to examine the physical nature of the parts of which the medium is composed, and the effect of changes of pressure occurring in one part on the pressure in other parts, and so to test our conception from this other point of view.

It is obvious the uterus cannot change its position, even to the slightest extent, without causing simultaneously corresponding changes, either in the positions or in the forms of the visceral parts abutting upon it—for the pelvis is full of viscera, all of which are pressed against each other and against the unyielding walls and resistant floor of its cavity. We have indeed already assumed that the visceral parts surrounding the uterus are deformable. A superficial acquaintance with these viscera teaches one of this; and the knowledge that the bladder and rectum are constantly undergoing changes in volume, shows the necessity for their easy change in form. Obviously, pressure upon the cervix by the finger, or traction upon it by forceps, can only cause the uterus to move by simultaneously displacing, and so deforming, adjacent opposing parts. So also, the rectum or bladder cannot distend unless the visceral parts pressed against them can be displaced according to the variations in volume of these

respective viscera. Of these, the uterus forms a relatively large and important part. It is closely related to both, but to a larger extent and more intimately to the bladder than to the rectum; and thus it is plain why displacements of the uterus should occur with change in volume of these organs, and should be influenced more by changes in the bladder than in the rectum. But, it is clear, such changes in the position of the uterus cannot occur unless a displacement or deformation of those other viscera, placed between the uterus and the pelvic wall towards which the uterus is moved, simultaneously happens; for it is on the possibility and occurrence of this also that the uterus is moved. The body of the uterus cannot be displaced upwards and backwards by distension of the bladder, unless the intestines above and behind it can readily be displaced —when the body of the uterus has grown so big, as by pregnancy or by fibroids, that these intestines are already displaced upwards, distension of the bladder can only occur by its own encroachment anteriorly into the abdomen. Nor, in the normal non-pregnant, can the cervix be caused to move backwards by the same change, unless the rectum can be deformed or displaced by the increasing pressure. Clearly, it is because the limits of such backward movement of the cervix are soon reached that the cervix becomes elevated. So also, for the same reason, when the uterus becomes pregnant, the cervix is raised posteriorly in the pelvis. Similarly, the cervix cannot be displaced forwards by distension of the rectum, unless the bladder is readily deformable or displaceable.

In considering the deformability of the visceral parts surrounding the uterus, it is first necessary to notice there is a considerable difference in this respect between the uterus itself in the non-pregnant state and surrounding organs. For the non-pregnant uterus is little deformable, the only frequent change in its shape, at least of any

importance or that can be perceived by palpation, is in the relative change in the positions of the body and cervix whereby flexions arise. It is for this reason that when changes in volume of surrounding organs occur, the uterus is bodily displaced; whereas the adjacent organs, displaced with the uterus, suffer much more change in shape because they are much more easily deformable. If we inquire into the cause of this difference, we shall see it is due to a difference in the states of contraction of the muscular walls of the respective viscera: that whilst the uterine musculature is usually tonically contracted, the musculature of surrounding organs is usually uncontracted, or if contracted, the contraction is very much less than that of the uterine musculature.

That the muscular walls of the bladder, vagina and rectum, except when the organs are engaged in active visceral processes, are uncontracted or little contracted, is shown above all else by the observation that for different postures, the pressures within them show corresponding differences; and that with movements of the individual, these pressures readily undergo similar—though not identical—variations. The change in pressure within the organs, due to change in posture, occurs immediately with the assumption of the new position: it occurs rapidly, with and as the new position is assumed; not slowly, as by a gradual adaptation to that position, such as we must suppose would be the case if the changes in pressure were caused by changes in contraction of the visceral walls. For owing to the nature of non-striped muscle tissue, characterised by the slowness of its contraction, such change of contraction, and so of resulting pressure, would be induced much more slowly than it actually is. And, clearly, during movement, as from one position to another, the much greater excursions of pressure, of variable intensity and rapid induction, which invariably occur, are

(for the same reason) independent of the contractions of visceral musculature—it will be admitted they are caused by variations of pressure of adjacent organs upon that in which the pressure is being observed. Clearly, the different pressures found in different postures are due to a change in the amount of the visceral thrust from the abdomen, itself dependent on the state of contraction of the thoracic diaphragm and abdominal wall, the tonic contractions of which are different for different postures : whilst the sequence of change, occurring during the movement from one posture to another, is the consequence of a series of changes of pressure occurring within the abdomen and transmitted to the pelvis, and can alone be due to the rapidly changing reactions of these body wall muscles—composed of striated tissue, characterised by the rapidity of its contraction—associated with the movement.

Moreover—when the rectum, vagina and bladder are empty or nearly so—the pressures within them, for any one posture, are very much about the same. The pressure in the bladder, as one would expect, is generally not so high as that in the rectum. That this should be so is to be explained by the lateral and anterior parts of the bladder being situated higher in the pelvis, nearer the abdomen ; whilst the part of the rectum in which the pressure is examined is situated in the depths of the pelvis. Moreover, these parts of the bladder are placed peripherally, closely applied to the pelvic walls, away from the direct line of the visceral thrust, and to these parts its contents are free to pass ; whilst the terminal part of the rectum is placed centrally, in the direct line of the visceral thrust. It is for this reason that, when movements occur, higher pressure values in the rectum are found than in the other organ.

But the pressure in these organs is not always even approximately the same for the same posture. The

accumulation of contents causes an increase of pressure within them which becomes greater as the volume of the contents increases. In a patient (Warren), eighteen years old, and in the seventh month of her second pregnancy, the pressure in the bladder was measured in various postures, and then after releasing 13 ounces of urine, much lower values for the same postures were found.

WARREN, EIGHTEEN YEARS OLD. — SECUNDI-GRAVIDA, SEVEN MONTHS PREGNANT; GIRTH OF ABDOMEN AT LEVEL OF UMBILICUS, 34 IN.

Position.	*Pressures* (all in mm. Hg.) in the		
	(1) Rectum.	(2) Bladder containing some urine—more than 13 ounces.	(3) Bladder after releasing 13 ounces of urine.
Left Side . .	2 to 10	10 to 12	8
Standing . .	23 to 24	30 to 32	24 to 26
*Stooping . .	14	34 to 36	30
Left Side . .	8 to 10	14 to 16	10

In this case, the pressures in the rectum were first observed; and then the pressures in the bladder before the release of urine, and finally again in the bladder after the release. Thus, the patient was made to assume the various postures in sequence three times, each series following closely upon the one before, and one cannot therefore be sure that the respective postures in the successive experiments were identical—which introduces an error; but the measurements of the pressure in the bladder before and after the release of urine are so different as to enable us to come to an opinion as to the effect of contents in organs on the pressures within these organs. So also, I

* The differences in the pressures in the bladder and rectum on stooping, is due to the fact that in this posture the tone of the belly wall is reduced, and the thrust into the pelvis diminished; but the pregnant uterus presses on the bladder in virtue of its weight more in this position, causing an increased pressure on the bladder.

found similar results in the following case, examined in the same way.

RAMSEY, EIGHTEEN YEARS OLD.—PRIMIGRAVIDA, SIX OR SEVEN MONTHS PREGNANT; GIRTH OF ABDOMEN, 35¾ IN.

Position.	*Pressures* (all in mm. Hg.) in the		
	(1) Rectum.	(2) Bladder containing more than 11 ounces of urine.	(3) Bladder after releasing 11 ounces of urine.
Left Side . .	8 to 10	14	8
Standing . .	30	32	20 to 24

At first sight, it would seem that the increase in pressure occurring with increase of visceral content is due to an increase in the tone of the non-striped musculature of the organ. Certainly, as a result of making many observations on the pressure in the rectum, the impression one gets is that higher values are usually obtained when the rectum contains fæces. The very considerable difference in the pressures found in the rectum and bladder taken simultaneously in the case of Jane Hall (see p. 174) was explained by the rectum containing soft fæces, the pressure of which, apparently, had induced a tonic contraction of the gut walls. For the pressures in the rectum in the left lateral (12 mm. Hg.) and supine (20 mm. Hg.) positions are unusually high—much higher than in the normal with the rectum empty. But although the muscular walls of the bladder may undergo a tonic contraction or an increase in tone with increase in bladder contents, it is plain the accompanying increase in pressure is not due alone to this. For, as we shall see, with increase of bladder volume and resulting increase of (intra-) bladder pressure, an increase in rectal pressure occurs. Similarly, with decrease in bladder volume, which results in a decrease in (intra-) bladder pressure, a decrease of the

pressure in the rectum simultaneously is observed. This shows at once that the increase in bladder pressure, consequent on increase of its contents, is not due alone to a contraction or an increased contraction of its musculature—for if so, the pressure in the rectum would be unaffected. Moreover, although the pressure within the pelvic viscera is greater when they contain contents than when they are empty, similar differences of pressure for different postures are still found. In the absence of tenesmus or straining, and apart from great distension of the viscera, it is still the posture of the individual—the tonic contraction of the abdominal wall and diaphragm—which is the great factor determining the pressures found. The greater this is, the less will be the effect of the tone of the visceral musculatures on the pressures within the respective viscera, and the more equal will the pressures in different viscera become; the less this is, as when the abdominal wall is weak or relaxed, the more prominent will be the tonic effect of the visceral musculatures, and the more effect will gravity —the weight of superimposed viscera—have in the pressure production; and so greater differences of pressure in adjacent as in distant organs may be expected, and in fact occur.* Thus it is that whilst gravity has little effect on the pressure production in the erect posture, because the body wall muscles are then well contracted, it becomes the predominant factor in the knee-chest or inverted positions, because then the tone of the belly wall muscles is considerably reduced (cf. footnote, p. 131). As we shall see, there is reason for believing that the tone of the muscular walls of the abdomino-pelvic cavity, and the tonic contraction of the musculatures of the hollow viscera are related: that when the former

* The case cited (that of Jane Hall) falls in the latter category, and more than ever explains the differences of pressure found in the rectum and bladder (see p. 174).

is good, as when the abdominal wall and diaphragm are well developed, the musculatures of quiescent hollow viscera are less contracted or are uncontracted; and, conversely, that when the body wall muscles are poorly developed and deficient in tone, the organs become much more contracted and consequently more muscular—as, for example, seems to happen in the case of the bladder and is perhaps a cause of the so-called fibrosis of the uterus—or, if the organs cannot respond, they dilate, as is instanced in the case of the stomach in enteroptosis.

Thus, although at first sight it appears the pressure within the pelvic viscera must be due to the tone of their own walls, and although under certain conditions such a tone is present and participates in the pressure production, we see—in the normal at least—it is not this which is the important factor at play, nor is this sufficient to mask the effect of the tonic contraction of the body wall muscles. Clearly, if the pressure within these organs was due in the main to a contraction of their own musculatures, or if their muscular walls participated in its production to any important extent, the pressure would be much more independent of the pressure outside the organs—that is, between them and surrounding parts; it would not be affected so much by posture as it is, nor would changes suddenly occur. Further, the pressures within the pelvic organs would not vary so precisely as they do in correspondence with small and distant changes of pressure—such, for instance, as are produced within the epigastrium by respiration; and, how could we explain the negative pressure found in the knee-chest and allied positions, if the visceral walls were usually contracted, or if their contraction was usually considerable?

And, if the contraction of the visceral walls was really great, it should induce a hardness or firmness of some sort of these walls, whereby they could be felt on examination—such for instance as is present normally in the uterus, but

which very rarely is found elsewhere to occur. It may be argued that the uterine walls are thicker than those of the other organs, and that is why they are felt. But the mere thickness of muscle is not sufficient to make it palpable. Uncontracted muscle is not hard; it is soft. Moreover, if such contraction were present in all the viscera simultaneously, and a more or less constant feature, as such hypothesis would have to allow; and if it were of so great an order as to produce within the organs pressures greater than those caused by the tone of the abdominal wall and thoracic diaphragm, the organs, besides being more or less rigid, would be spherical or cylindrical, their surfaces would always be curved, and adjacent viscera could not be insinuated into clefts between others. Thus, spaces between organs would exist in which fluid would collect; and the return of venous blood from the viscera would be interfered with and impeded. But we know that under natural conditions, and when in health, the pelvic viscera, other than the uterus, are so soft as to be imperceptible even on bimanual examination; nor does fluid exist in any quantity between them.

So far, then, from believing that the visceral walls are usually contracted, such considerations coerce us to believe the reverse is actually the case; that apart from visceral acts, the visceral walls are uncontracted; or, if contracted, their contraction when compared with the force exerted and the pressure produced by the healthy and normally contracting abdominal wall and thoracic diaphragm is inconsiderable, and thus may be neglected. They coerce us to believe that all the viscera are pressed—willy-nilly—against each other and against the walls and floor of the pelvic cavity by the visceral thrust from the abdomen; whereby the organs come to possess certain more or less definite and peculiar shapes, opposing surfaces being flattened by the pressure within and communicated to them, with the

production of facets as fleeting as the causes which produce them; and whereby parts of other adjacent organs are squeezed into clefts between opposing organs, so that, on section of the pelvis, the viscera are revealed all closely packed together within the cavity containing them.

2. Concerning the Functions of the Female Pelvic Viscera in respect to the Contraction of their Musculatures

Nor, when we come to consider the functions of these organs, is there any reason to suppose their musculatures, when the organs are empty, are usually contracted. As contents collect, fleeting contractions probably appear, occurring rhythmically, like those of the spleen. These presumably become gradually greater and last longer as repletion is approached, ultimately giving way to that contraction which determines an emptying of the organ. It is the increase in contraction of the visceral musculature and the increase in pressure within the viscus so caused, that gives rise to sensations which manifest to the individual the necessity of emptying the organ. Usually these sensations only arise when distension has become considerable, but they may do so when the organ contains but a small amount of contents—notably so in the case of the bladder. Sometimes the desire to empty the bladder recurs soon after the organ has been emptied, when one is sure but little urine is within. If this desire be resisted, it passes away; whilst if yielded to, but a few drops of urine are passed. The explanation of this can only be that the bladder in such cases is contracted; and that the contraction gradually fades away. And, according to Hart and Barbour, the empty adult bladder occasionally is found "oval in shape, corrugated and firm to the touch"—because, as they say, "it has been caught in

systole," i.e. when it was contracted [35]. In such cases, the bladder lumen, on sagittal section, is more or less vertical and forms a continuous slit with that of the urethra, and is therefore quite unlike that usually described; and the organ presents as a definite lump in the pelvis, which presumably can be felt on bimanual palpation. But this, though an interesting, is a rare condition. I have not found it mentioned in the present-day textbooks I have referred to; nor have I come across it in my clinical experience. Such a bladder, however, is depicted by Cameron [14] (see Fig. 13, p. 59); but the condition is abnormal and probably associated with a diminished intra-abdominal pressure—such, for instance, as obtains in enteroptosis.* But even though in the normal the bladder should always have to contract to this degree for micturition, so that it is oval and firm when empty, which, however, cannot be allowed,† we must believe that such contraction soon fades away—that the wall of the empty quiescent organ is uncontracted.

And, it would seem, that as urine collects, but little contraction occurs. Even when the bladder contains a moderate quantity of urine "it is still so flabby that it cannot be palpated" (Winter and Ruge [105]). And since the bladder is continuously being pressed upon by surrounding organs, compressed between them and the superficies of the pelvis, it is evident a greatly tonically contracted musculature, which would cause the pressure in the bladder to be maintained considerably

* A similar bladder is depicted by Elliot Smith in the *Journal of Anatomy* for 1908, the drawing being made from a section of a pelvis of a Soudanese negro. In a note the author says: "This infantile type of bladder—a torpedo-shaped organ projecting well above the pubes, even when empty—seems to be very common in Soudanese negroes" [93].

† "When empty, the bladder is felt to be soft and flabby" (Winter and Ruge [105]).

raised above the pressure between it and surrounding parts, would not only be unnecessary, but even undesirable. Were such the case, not only would it render the entry of urine into the bladder more difficult, necessitating a greater expenditure of energy on the part of the ureteric musculatures and on the secretory cells of the kidneys, possibly damaging them,* but it would make more difficult the retention of urine in the bladder, requiring more energy on the part of the sphincters.†

Moreover, since the urine is a mobile liquid, and the obstacles to its discharge are but the resistance of these sphincters and the negligible frictional resistance of the (female) urethral walls; and further, since the pressure of surrounding organs on the bladder can suffice to cause the escape of urine when the sphincters relax, or when they are dilated (as by catheter) or have become inefficient (as in some cases of prolapse and atonia of the pelvic floor); it seems that a contraction of any magnitude of its own musculature is scarcely necessary for micturition. It may be necessary to induce relaxation of the internal sphincter, acting not so much directly as by setting in motion a reflex nervous mechanism; and to cause the desire and so lead to the act of micturition. The gradually increasing stretching of the bladder walls, caused by the continued accumulation of urine, not only directly excites the bladder wall to contract, but sets up sensory impressions or impulses which pass up to the lumbar cord and are reflected down again by efferent nerves to the (non-striped) sphincter causing its inhibition. But the

* The effect of phimosis and of stricture of the urethra on the kidneys, in causing bilateral hydronephrosis, and ultimately such atrophy of the organs that uræmia results, is well known.

† These are the internal (non-striped) surrounding the neck and trigone of the bladder; and the external, the uro-genital sphincter (striated), embracing the urethra and in part the vagina (see Kalischer)[49].

disturbance of the lumbar centres causes other impulses to arise, which being transmitted to the brain, acquaint the individual with the necessity and thus cause the desire to micturate. But we can scarcely believe when the bladder is emptied by catheter, the patient lying quietly on the side, or in the lithotomy position under anæsthesia, that the bladder wall contracts, much less that the bladder is emptied by such contraction. Under these circumstances, pressure upon the hypogastrium is generally necessary to ensure complete emptying of the organ; and from this it may be argued that contraction of the bladder wall is necessary under ordinary conditions for the complete discharge of urine. But in these positions the intra-abdominal pressure is low, whilst in the upright or crouching posture it is very much higher, and this clearly must facilitate the act, and render the contraction of the bladder wall less necessary than it otherwise would be.

When the intra-abdominal pressure is lower than normal (or when an external resistance greater than normal has to be overcome, e.g. in phimosis), contraction of the bladder for micturition is much more likely to occur. Such seems to be the reason that the bladder is such a muscular organ in the lower animals and in the human fœtus, in which the oval shape is always found (Hart and Barbour [35]). For in the former, the intra-abdominal pressure is obviously less than in orthograde man; whilst in the latter, expulsion of the urine, which occurs into the liquor amnii, is only possible by overcoming the pressure to which that liquid is subjected. When we remember the anatomical and physical relations of the human adult bladder, the saucer-shaped form of the normal empty organ; how its extensive base is apposed to and pressed down on the floor of the pelvis to which it is attached (however loosely) by connective tissue; and how its posterior duplication, medianly, is attached to the cervix and vagina; one can

scarcely believe that the bladder, though spherical or ovoid when distended, remains so in virtue of its contraction as micturition proceeds and is completed. For such would obviously require and cause considerable dislocations of its parts from apposing parts, with a loosening of its attachments. Especially would its connection with the vagina and cervix be much less close—as loose, indeed, as is the connection of the rectum with the vagina posteriorly. But the conditions of these two viscera in this respect are very different. The firm adhesion of the bladder posteriorly to the cervix—by fatless connective tissue *—is, to my thinking, a reason for believing that as soon as the internal sphincter of the bladder is overcome and micturition started, the contraction of the bladder dies away, the act being continued by the intra-abdominal pressure—if not entirely, then at least in very great part—so that instead of remaining spherical, as it is when distended, it becomes flattened from above downwards, assuming its usual concavo-convex form.

In the case of the rectum, there is much more necessity for a contraction of its musculature during defæcation; because the contents to be ejected form a semi-solid mass. Not only is the pressure of surrounding parts on the rectum in the resting state insufficient to empty the rectum, but even the much greater pressure caused by straining alone does not as a rule suffice. When the contents of the rectum are fluid, straining or even a cough may cause some escape; and even when semi-solid, great straining alone may result in an ejection, but this is rarely complete, and the force to be used is out of all proportion to the effect. Peristalsis is essential for the efficient performance of defæcation. This difference in the reactions of the bladder and rectum, necessary for the function of each, is probably the explanation of the closer relations of the bladder than

* See footnote, p. 73.

of the rectum to the genital tract. The rectum is more free. According to Paterson [78], it traverses a special tract in the pelvis. The region it occupies is distinguished from that occupied by the bladder by the greater looseness of its connective tissue—conspicuous for its fat—which surrounds it, and which anteriorly is separated from the cervix and the upper part of the vagina by Douglas' pouch. By this separation, not only can its distension and change in volume easily occur—a provision, however, unnecessary for such change—but vigorous peristaltic waves can easily pass along it, the necessity for which seems to have determined the peculiarities referred to. And this is the reason why, as the gut passes through the pelvic floor aperture, its non-striped muscle strands interweave with the striated muscle fibres of the pubo-rectalis. Obviously, without such adhesion, peristaltic waves instead of causing expulsion of the fæcal mass would result in the gut itself being withdrawn upwards over its contents without these being passed per anum. But although peristalsis, in the case of the rectum, is necessary for defæcation, and although, when fæcal contents are present within it, some contraction of its walls may exist, there is no reason for supposing that when empty its walls are contracted.

The vagina possesses a well-marked non-striped musculature—not distinctly separable into strata, but composed chiefly of fibres internally circular and externally longitudinal (Quain [83]). But it is plain, this musculature is usually uncontracted; or, if contracted, that the force of its contraction is not sufficient to prevent the easy deformability of the viscus and its adaptability to the parts about it. As we have seen, it is not the musculature, in virtue of its contraction, which has given to the vagina its peculiar form—otherwise the organ would be cylindrical

—like a Ferguson's speculum, as all the writers before Boivin and Duges (1833), and even they to some extent, believed.* But just as in the case of the empty bladder, the reason its opposing walls are in apposition is that they are pressed together by the pressure of viscera above and the resistance of the pelvic floor below. The intra-abdominal pressure and its excursions, which have caused the transition from the infant to the adult type, and the spreading of the vagina backwards in the pelvis, explains the flattening of its walls from above downwards, that is the spreading of the organ from side to side: they are the responsible factors determining the shape of the organ and its position in the pelvis. These peculiarities show that the intra-abdominal pressure is a force out of all proportion greater and more important than the force caused by the tone of the vaginal walls themselves.

That the vagina is easily deformable is obvious on examination. Its anterior wall can be pushed upwards and forwards by a finger in the vagina; and by this means the upper internal aspect of the pubic arch easily palpated. The same, in a reverse direction, is evident on manipulating with a sound in the bladder. So also the lateral walls of the pelvic cavity can be explored per vaginam. Thus, the tendinous "white line" can often be felt; and the ischial spines clearly. The same is the case with the posterior vaginal wall: a finger in the rectum can bulge it readily forwards—by which means indeed the pubic arch is easily defined. Similarly, the upper part of the vagina can be invaginated by pulling the cervix downwards by forceps. Such invagination, clearly, is conditioned by an absence or a negligible amount of tone in the vaginal walls.

Thus, it is not surprising that the vagina, which serves merely the purpose of a passage between the surface of the

* See Mauriceau [62], Levret [57], Denman [18] and others.

body and the uterus, is readily dilatable. It is a passage wherein spermatozoa can be deposited and so more easily and certainly reach the uterus, and by which, after fruition of the ovum, its perfected parts can be extruded. In this, the vagina plays an important part. It not only directs the fœtus to the surface of the body, furnishing a mode of escape; but being adherent to the pelvic floor aperture below (pubo-rectalis) and structurally continuous with the cervix above,* it ensures expulsion of the child as the result of the uterine contractions. Thus, it prevents the uterus withdrawing itself upwards over the child without this being born—such as obtains when rupture of the lower uterine segment occurs and the fœtus is forced into the peritoneal sac. To what extent the longitudinal muscle fibres of the vagina participate in this, it is difficult to say; nor do we know the rôle of the transverse fibres when the organ is dilated. Just as the longitudinal fibres probably come into play during parturition when the vagina is being extended, so probably the transverse fibres contract during its dilatation. But they can scarcely limit its dilatation. This depends more on the volume of the body being extruded through it or passed into it and on the size of the pelvic cavity and displaceability of the adjacent visceral parts than on a contraction of the vaginal musculature. Apart from such factors, the dilatability of the vagina seems to depend less on the non-striped muscle in its wall, than on the elasticity of all the tissues composing its wall, the loss of which explains the diminished dilatability in the old. In women during the child-bearing age—that is, during their prime, when all the tissues are supple—and in those in whom the hymen has been well ruptured, it is only just beyond the vaginal orifice, where the vagina

* Before the time of Fallopius, the vagina and uterus were generically designated "the Womb," the vaginal orifice being referred to as the "Os Externum."

with the urethra and rectum pass through the pelvic floor aperture, that any constriction of the vagina occurs. It is only here that an obstacle to the insertion of specula or of pessaries of ordinary size is met with; and it is this which has to be overcome in the extrusion of the fœtus or the removal of pessaries from the passage.

It has long been thought that the tone of the vagina is the effective means whereby pessaries are retained, and the reason they are efficacious in preserving the corrected position of a misplaced uterus. So also, as we have noticed, a rôle has been assigned to the vagina in the maintenance of the uterus in its normal position on this account: in virtue of its tone, the vagina has been supposed to possess a rigidity, and acting like a stem, to support the uterus or at least to prevent the advance of the cervix to the vulva. But such notions are erroneous. The vaginal musculature has no tone; or the force resulting from its tone is inconsiderable, and of negligible importance in these connections—it is not comparable with the force exerted by the abdominal wall and diaphragm. But when distended, some such tone may appear. But, in parous women certainly, it is not sufficient to embrace bodies inserted into it firmly, unless these cause as well a stretching of the other tissues in its wall, and the elasticity of all comes into play. Even during coitus, the contraction of the vaginal musculature, if indeed it be contracted at all, is insignificant. The phallus is embraced firmly in the lower part of the vagina by the pubo-rectalis muscle, and pressed against the lower sharp border of the pubic arch by this [13], as are the fingers often during internal examination; but beyond this sphincter, the pressure on the body is due more to the intra-abdominal pressure than to the reaction of the vaginal musculature. The irregular, curved and misshapen forms, into which the wax cylinders, used by Dickinson to confirm Budin's experiments, were converted,

show this is so [20]. The increased pressure of the visceral parts about the vagina, combined with the increased contraction of the pubo-rectalis, produced by the patient "drawing-up the parts," explains the deformation of the wax cylinders introduced into the vagina. During this remarkable act,

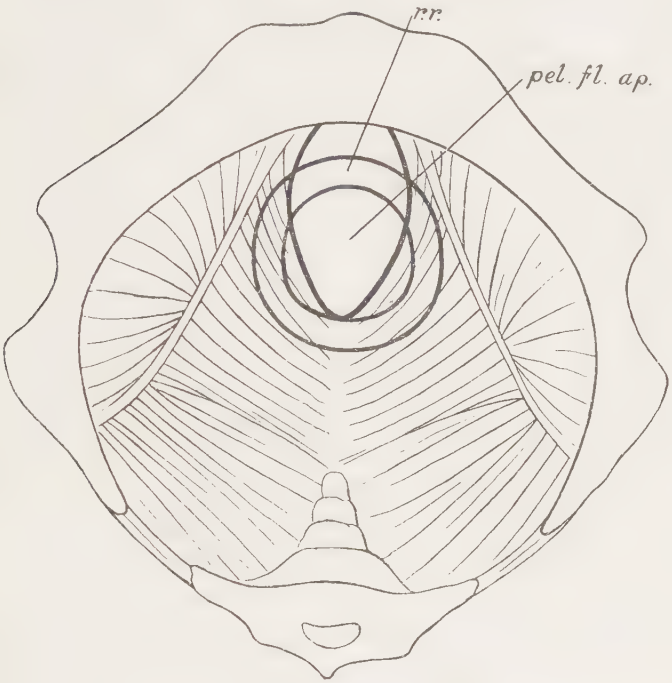

FIG. 18.—REPRESENTING THE MECHANISM BY WHICH PESSARIES ARE RETAINED.

The pessary bulges the vaginal walls out laterally, and comes to rest through these on the lateral arms of origin of the pubo-coccygei. After Halban and Tandler.

r.r. = rubber ring. pel. fl. ap. = pelvic floor aperture.

the instinct for which in relation to the preservation of the species is as obscure as its origin is remote, and which affords an interesting spectacle to students of philosophy, the glans and the cervix maintain more or less their approximated positions: the cervix is elevated and the vagina elongated

by the thrust of the male—which is known on occasion to have perforated the posterior vaginal fornix [11]; whilst, on recession of the glans, the cervix descends, being pressed downwards from its elevated position by the intra-abdominal pressure in the female—the vagina playing very little if any part in this or in embracing the organ of the male. Nor are pessaries retained within the pelvis by a tone of the musculature in the vaginal walls; but, as Halban and Tandler have shown [34], and any one by examining the living patient can convince himself, by the pelvic floor. The lateral arms of the pessary *in situ* thrust the vaginal walls out laterally, beyond the lateral limits of the pelvic floor aperture; and thus, they come to rest on the upper borders or surface of this musculature, which form the lateral boundaries of that aperture (Fig. 18). It is for this reason that pessaries shaped as rings are so commonly employed—they are most satisfactory: it is found they are much more serviceable than narrow instruments.

That the little deformability of the uterus, in the non-pregnant, is due to its tonic contraction, is shown, I think, by the remarkable change which occurs when it becomes pregnant. Instead of being firm, it becomes soft; and so soft that it is often unperceived on palpation, or mistaken for the distended bladder. Clearly, the uterus when pregnant is often uncontracted; indeed, until Braxton Hicks showed that contractions occur [38], the pregnant uterus was thought to be always relaxed. But though conspicuous for its contractions, these are infrequent and of short duration. And in the intervals between these contractions, the uterus lies flaccid, taking on the form imparted to it by the pressure of surrounding parts (e.g. of the lumbar lordosis and abdominal wall), and is irregular in shape in consequence of the fœtal form and position. The uncontracted organ, plainly, is highly deformable, to

the extent indeed that the pressure within it must be equal to that between it and adjacent organs; and thus does the uterus come into line with the other pelvic viscera when these are quiescent and not engaged in visceral acts. Occasionally, too, as after abortion, the uterus loses its firmness, becoming boggy in feel; and sometimes, even apart from pregnancy, so lax that it can easily be pressed out of shape.

It may be urged that the firmness of the non-pregnant organ is due to its connective tissue, in support of which may be adduced the increased rigidity in multiparæ, and especially in the condition of so-called fibrosis. This, however, is not my opinion. Histological examination of the uterus shows its wall contains much more muscle than connective tissue, that muscle is the preponderating element. And the bladder wall, size for size, or thickness for thickness, contains perhaps quite as much connective tissue; and connective tissue, moreover, is easily deformable. But a tonically contracted muscular organ, such as the contracted heart, the contracted biceps, even the contracted bladder, is not easily deformable.

Nor, in my opinion, is there a relative increase of connective tissue in the uterus in multiparæ. In the course of my duties as Pathologist at the Hospital for Women, I have examined histologically, with great interest and care, a good many uteri removed from multiparæ in the hospital for so-called fibrosis—that is to say, from women past middle age, who presented the clinical picture of increased, perhaps profuse menstrual loss, and revealing on examination a uterus a little larger and firmer than usual, but otherwise apparently normal. In none of these cases have I found a relative increase of connective tissue; in all, I found the increase in size to be due to an increase of both muscle and connective tissue, of which the increase in muscle was the more striking and the dominant feature.

This is contrary to current teaching*; but that it is so, does not make me reject the evidence of my senses. This opinion, moreover, is supported by the observation of Shaw [91], who, working on this subject, came to the same conclusion—that these uteri, said to be fibrotic, show no disproportionate increase in connective tissue, but present all the appearances of hypertrophy; and that they are in truth hypertrophied, at least as far as their muscular walls are concerned.

It is said the reason the uterus is a muscular organ is that it has to eject the fœtus at term; and it is supposed that this is the sole function of the uterine musculature. This explanation puts me in mind of a little story, about a candidate up for examination, told by a lecturer to his students when irritated by their inattention and joking. Asked why the cells lining the uterine mucous membrane possessed cilia, the candidate replied they were for the purpose of child-birth, to waft the infant into the world—in which case, the lecturer told us, the smile was not with the student. When I reflect on the thickness of the musculature of the non-pregnant uterus, and remember that by far the greater part is homologous with the muscularis mucosæ of other viscera [104], and that in no viscus is this part associated with movement of the contents within the viscus, but in all with the regulation of the blood supply to or return from its glandular epithelium; and when I recall how thin the whole musculature of the uterus becomes in pregnancy, I cannot but reject the idea that the musculature of the non-pregnant organ is for a cause that may never arise, I cannot but think that it has some other purpose, such as its homologues have elsewhere, namely the regulation of blood to the endometrium necessary for some reproductive or perhaps other end. This opinion

* It is admitted by Eden and Lockyer that some cases of so-called fibrosis uteri are really cases of hypertrophy of the uterus [26].

harmonises with the well-known monthly cycle the endometrium undergoes, culminating in menstruation, so often preceded or accompanied by painful uterine contractions, occurring before or with the rupture of the blood capillaries in the mucosa and the escape of blood into its tissues and into the uterine cavity—not associated with or caused by the discharge of blood from the uterine cavity into the vagina. Thus, in the so-called fibrosis of the uterus, the hypertrophy of the musculature observed on microscopic examination, associated as it is with an increased menstrual loss, has for me a special interest.

3. The Influence of the Peritoneum and the Contents of the Hollow Viscera

Whilst it is clear the easy deformability of the viscera surrounding the uterus, upon which the mobility of the uterus must greatly depend, is due in great measure to the little contraction of their muscular walls, other conditions conform to the same end. The existence of the peritoneal sac, and the presence of contents more or less mobile in the hollow organs both play a part in facilitating uterine mobility by allowing displacements of visceral parts one on another to occur.

All the viscera surrounding the uterus are partially, and some almost completely covered with peritoneum, the invaginations of which dip down from the abdomen into the pelvis, separating to some extent adjacent viscera. The existence of these extensive capillary-like clefts clearly permits the easy gliding of opposing visceral surfaces over each other, allowing changes in position or in form of the respective viscera to occur. For instance, in virtue of the utero-vesical cleft, the opposing surfaces of the uterus and bladder can so glide on each other that the uterine body

can move from side to side in response to lateral pressure changes, as variations in volume of the pelvic colon or upper part of rectum may cause. Similarly, the bladder itself can easily distend, displacing surrounding organs, and its walls stretch to accommodate the urine. For as the bladder distends, the uterus is not only raised and displaced backwards, but changes in the relative positions of the adjacent uterine and vesical walls occur. So also, by the same movement, the visceral parts above and behind the uterus are displaced upwards into the abdomen—a change in their position easily effected; for they slip upwards, in virtue of the peritoneal cleft between them and the uterus, in response to the increasing volume of the pelvic viscera, and the increase of pelvic pressure which such change in volume produces. Similarly, in virtue of Douglas' pouch, the cervix with the posterior duplication of the bladder, can move upwards and downwards on the rectum—visceral impactions from above, the result, for instance, of cough, causing their descent; whilst with the cessation of such impactions the cervix returns to its ordinary position.

In the same way, it is plain, the opposite walls of hollow viscera may be displaced in a gliding fashion over each other—except when the viscera are distended or their musculatures so contracted—as in the non-pregnant uterus —as to prevent such movements. Such obviously can occur in the case of the bladder, the wall of which is so soft as to be impalpable bimanually, and its musculature so rarely or so little contracted in the adult that it takes the form of a concavo-convex body in adaptation to surrounding parts. Thus, we may reasonably suppose gliding movements of its upper wall with the superimposed uterus often occur over the lower wall, from which it is separated by a layer of urine; that such, for instance, especially happens when sudden pressure waves, deflected from the hypogas-

trium, pass backwards over these viscera. Further—when the bladder contains an appreciable amount of urine, but is not greatly distended, other changes in its form can easily occur; for instance, recession of its superior surface medianly, and bulging of its lateral parts to the sides and upwards in compensation—a sort of natural fluctuation can easily be produced. Clearly, the mobility of its contents permits this change in form, which results from variations of pressure about the organ. The same argument may also be applied to the pregnant uterus, when liquor amnii has appeared to an appreciable amount, and the uterine wall is uncontracted. So also the rectum, when it contains fæcal material, adapts itself to the pressure about it, except when its contents are hardened by the absorption of water and form scybalous masses.

But when these organs become distended, these conditions do not obtain or not to the same extent. The stretching of the visceral walls acts as a stimulus causing contraction of their musculatures; and this, when considerable, causes the organ to become for the time being indeformable. Apart from such muscular activity, distension brings the elasticity of the other tissues into play, and interferes considerably with the deformability of the viscus. Distension sufficient to produce this change is associated with a considerable rise of pressure within the organ; and causes a diminution in the mobility of the uterus. Thus, the uterus is less mobile when the bladder is distended than when it is empty. The same change is not seen to the same extent in the case of the rectum; for even when ordinarily distended, the space of the pelvis it occupies is small, much smaller than that of the distended bladder, and so the effect of its distension on the uterine mobility is correspondingly less than that of distension of the bladder. In the same way, the presence of tumours interferes with the uterine mobility—such, for

instance, as ovarian cysts. These are not readily or at all deformable, because of the pressure within them; and, like distension of the viscera, by taking up a certain amount of the available space in the pelvis, obviously must limit the movements of the uterus.

4. The Connective Tissue in the Pelvis and the Tissue Fluids in relation to the Uterine Mobility

Whilst the viscera surrounding the uterus, being for the most part little contracted or uncontracted, are free to vary in volume, and thus to change the relative positions of their parts to those of adjacent parts, and to change in form, the volume being constant, only coils of intestine—the pelvic colon and small gut—possessing relatively long mesenteries, are able to be bodily displaced to other parts of the abdomino-pelvic cavity. But such displacement of the other organs in the pelvis is impossible. For their lower extremities, or the passages which pass from them, as we have noticed, are adherent to the margins of the pelvic floor aperture—the non-striped fibres of the visceral canals interweaving with the horizontally disposed fibres of the pubo-rectalis [36, 49]. And further, the connective tissue, which forms a matrix and envelope for them all, in which too the centrally placed uterus and the invaginations of the peritoneal sac are embedded, is adherent to the walls and floor of the pelvic cavity, not only by means of the thickenings—the so-called ligaments—within it, but in the whole extent of its superficies. Dissection easily discloses this; it is rendered vivid to the pathologist engaged in removing the pelvic viscera in mass *post mortem*. Thus, all the visceral parts, since they are adherent to adjacent visceral parts by means of this connective tissue, except where the peritoneum intervenes,

are indirectly connected with the walls and floor of the pelvic cavity. It is obvious that the connection between adjacent visceral parts and that between them and the limiting wall and floor of the pelvis must tend to limit the movements of the viscera—one would think this connective tissue (not the thickenings only, but all of it) would cause fixation of all the organs, such as it does after the pelvic visceral mass has been removed and hardened; but we know that in the living this connective tissue only limits wide movements, such as do not occur within the normal range of movements in the body. The normal mobility of the uterus shows that the constitution of this connective tissue is such that it does not limit normal movements of the cervix, such as variations in volume of surrounding viscera produce, or which are imparted to it and to the other pelvic viscera by sudden rises of pressure produced within the abdomen. Whilst we can readily understand how such organs as the intestines or the Fallopian tubes and the body of the uterus, should be readily mobile, because they are so extensively covered with peritoneum, in the case of the cervix the conditions that obtain in this respect do not suffice to explain its mobility; for only the posterior surface of its supra-vaginal part is covered by peritoneum, and so separates it from the rectum. How, then, we may ask, is the cervix mobile? A closer analysis of the "medium" should disclose the conditions.

The collection of tissues we have described as this medium consists of cells and cell-processes, of fluids between the cells, and of visceral contents enclosed within collections of the cells, which contents are all more or less mobile. The cells of which the medium is largely composed are all alive! And they possess a form which, though more or less typical for the variety of the cell, corresponding with the

work it has to do,* and with its position,† is not absolutely constant. Variations normally occur in which at least two opposing factors—the pressure within the cell and the pressure without it—participate. On the one hand, the intracellular tension of the cells, which varies with their activity and metabolism, tends to cause them to assume the spherical shape (the result presumably of osmotic pressure or surface tension), or by interactions between different parts of their protoplasm to maintain the form they have inherited ‡: and, on the other hand, the pressure about the cells, that caused by adjacent cells pressing upon them, as in epithelial tissues; or the traction of contiguous cells, as in muscle, and in a less degree in connective tissue, tend to prevent the assumption of the spherical shape, or to deform the shape inherited. Thus, he cells are elastic: they can be deformed by an extra pressure without them, or traction upon them, which they resist; and they tend to resume and do resume their usual form when this extra pressure or tension is removed —doing so as long as they are alive, as long as they can control or influence in any way their immediate environment, as long as they can draw upon an adequate supply of blood.

But just as there is considerable difference in the functional activities, and the corresponding intensities of metabolism, of different types of cell, so also we must believe there is a difference in the response of these different types to environmental forces of the same magnitude (pressure and traction).

* For example, epithelial cells, non-striped muscle cells, and connective tissue cells.

† For example, as in the difference in form of the epithelial cells of the bladder—those next the bladder lumen being more or less flattened, those next the muscle wall more columnar.

‡ Compare, for example, the form of such a unicellular organism as Paramœcium [17].

The same degree of pressure or of traction will presumably affect cells in which the average metabolism is low more easily than cells in which the average metabolism is higher. Connective tissue cells must therefore be more easily affected by changes of pressure and of tension than epithelial cells, and be less easily able to maintain their original form than epithelial cells. (When we compare connective tissue cells with striated muscle cells in this respect, what a difference do we see in their reactions to such forces!) Thus, when changes of pressure affect the conglomeration of tissues in the pelvis, displacements of parts on each other, separated by collections of connective tissue cells, should presumably occur more readily than displacements of parts of the same tissue or organ on each other—that is, of parts not separated by connective tissue but united by epithelial cells, or indeed by mesoblastic cells of a higher order than connective tissue cells, for example, of non-striped muscle cells.*

But though the medium consists largely of cells, other parts enter into its composition. Innumerable spaces, containing fluid (lymph), partially separate the cells and link up to form lymphatics, into which some of the products of the cells' activities are pressed.† A remarkable network of intercommunicating channels permeate especially the mesoblastic tissues, which form so large a content of

* This perhaps explains how it is collections of specific cells comprising organs have come to be separated from other specific cells by connective tissue.

† The amount of lymph in the cell spaces depends upon its production, the result of the metabolism of the cells (and therefore depends on the type of cell) and the interchange between the cells and the chemical products in the blood stream: and on its removal, which occurs by way of the blood stream or by the lymphatics. The entry of the lymph at its source into the blood depends on the difference in osmotic pressure between the lymph and blood: the passage into the lymphatics results from the cellular pressure—that is, the intra-abdomino-pelvic pressure at this level

the pelvic visceral mass. These channels are so thin that on cross section, even under the microscope their unicellular walls are scarcely visible, or are only visible on account of the nuclei bulging the cell walls generally inwards towards the lumen of the capillary. Through these, streams of blood are passing, almost ceaseless in their continuance, supplied by the high-pressure blood in the arterioles, and pouring into the veins.

This separation of cells and their processes from each other by cell spaces and capillaries of varying states of distension, occupied by easily displaceable liquids, must act like minute water-beds only semi-distended, readily permitting movements of cells on each other; and although such individual movements may be slight, collectively they are effective in allowing appreciable changes in position of parts related to each other. The displaceability especially of the blood to other parts of the vascular system, such as caused by sudden rises of intra-abdominal pressure, as by cough, will clearly facilitate such movements; for under such conditions, the force of the visceral impaction becomes temporarily greater than the blood pressure in the smaller arterioles, and thus the blood is squeezed out of the capillaries into the veins supplied by them, and the capillaries temporarily obliterated. The veins themselves are similarly affected: variations in their volume caused by variations of pressure outside them will clearly facilitate movements of surrounding parts.

That the blood and lymph content of the tissues does form an important factor in the mobility of the organs is shown by the suppleness of the normal and the fixity of the organs in inflammatory diseases. The distension of the cell spaces with accumulated lymph and leucocytes, and the stasis of the blood in the congested capillaries, produce, as we know, hard and fixed lumps, often difficult or impossible to distinguish from visceral parts or tumours.

These changes we know do affect the tissues of the organs themselves, but the greatest changes occur when the connective tissue in which the organs are embedded is also inflamed. This combined with the fact that the enveloping connective tissue, that situated peripherally between the collective viscera and the walls and floor of the pelvis, is loose (that is, contains many cell spaces) and in obese women is fatty; that it yields more easily than epithelial and other mesoblastic cells to pressure changes; and that in these peripheral regions the condensations of the connective tissue—which form strands medianly—fray out and disperse, so that they are scarcely to be found or identified; lead us to the opinion that the condition or structure of this connective tissue plays a large part in allowing mobility of the organs, especially of the cervix.

Thus, though the cellular components of the visceral mass, being composed of that semi-fluid stuff—protoplasm—which when alive, in virtue of its potential energy, affects the environment by assuming or tending to assume definite forms, would of themselves comprise a highly viscous liquid or semi-solid mass of very marked viscosity, and perhaps not easily deformable; in consequence of being collected into organs, separated from each other by invaginations of the peritoneum or by strata of connective tissue; many of which organs are hollow, and conspicuous during resting conditions for the little contraction of their muscular walls and often for the mobility of their contents; and moreover, in consequence of the whole being permeated by lymph spaces and blood capillaries, not usually in a state of turgescence, continuous with lymphatics and veins respectively, the presence of which are also of importance; we have a condition of structure whereby changes in position or of form of the different parts of the visceral mass can fairly easily occur. Thus, besides the physical character of plasticity, whereby individual parts

will give way before other parts according to differences of pressure, the conglomeration of pelvic tissues possesses also the physical character of ready displaceability of its units, one upon another, within certain limits, which renders it comparable much more to a liquid possessing a certain mobility than the nature of the cellular components of the mass by themselves would allow us to suppose. This, whilst very different from the free mobility of the molecules of a mobile liquid—in that in the former the displaceability of the units is restricted, whilst in the latter it is unlimited—is such as to readily explain the uterine mobility. Much more intelligible does this mobility become when we recognise it occurs as the result of changes of pressure occurring in the tissues about the uterus.

5. The Incompressibility of the Medium

But there is another peculiarity of this medium to notice: viz. its incompressibility. Whilst the position of the uterus depends on the volume of surrounding viscera, and the support rendered to all by the walls and floor of the pelvis, it does so because these surrounding viscera, especially those below it—the bladder, vagina, and often the rectum—are incompressible. So also the change in the position of the uterus, consequent on changes in the volumes of surrounding viscera, although clearly dependent on the deformability and displaceability of the visceral parts about the uterus, is determined by the increase of incompressible material in the pelvis to one or other side or below it—the certainty of its occurrence depends on this. Not only these physiological displacements of the uterus, but also that occurring during the early growth of the individual and the pathological displacements as well, become much more easily intelligible—indeed, I might say, they only become intelligible—when we recognise this

really very important physical feature of the visceral mass: that however much the visceral mass, which completely fills the abdomino-pelvic cavity, is compressed, provided none of it escape from the cavity, it cannot get smaller. This, at least, applies to the great bulk of the visceral mass, whether abdominal or pelvic; for only in the case of the gas contained within the intestinal tract, normally of small volume, can such diminution occur. Other than this factor, which can only affect the parts behind the uterus (rectum) and those above and behind it (intestines), the volume of the visceral mass can only be diminished by the discharge of visceral contents to the exterior—prevented in the absence of visceral acts by the presence and action of sphincteric mechanisms; or by displacement of blood in the veins and of lymph in the lymphatics into the thorax—a displacement which, so far from being prevented, is constantly occurring, and is an important effect of the pressure within the abdomen.

We have referred often enough in the previous pages to this incompressibility. We have but to think of the material substances of which the visceral mass is composed to admit that this mass for the most part is incompressible. Clearly, the tissue cells and their processes, the visceral contents (except gas), and the lymph and the blood, are all incompressible. Owing to this incompressibility, it is clear that as long as the visceral contents remain constant, that is, as long as they are neither added to nor ejected, nor displaced to other visceral parts, as from the colon to the rectum, though the shape of the viscus may be altered by change of pressure about it, its volume remains the same, or practically the same. It may be, and probably is, diminished to some small extent, by the squeezing out from the visceral walls of tissue fluids and blood to the lymphatics and veins outside the viscus, when the visceral wall itself contracts, and so causes the pressure within the viscus on its contents,

as also on the tissue fluids and blood within its walls, to become greater than the pressure immediately outside the viscus. But this factor cannot very materially affect the volume of the viscus; for the blood is renewed from the arterioles, unless the pressure in the visceral wall becomes greater than the blood pressure in the supplying arterioles, in which case the visceral tissue must become blanched. When we reflect that the connective tissue between the viscera and between them and the walls and floor of the pelvis are subjected to the same pressure as the viscera themelves (except when they contract), and that the viscera about the uterus are deformable, it is obvious that the tissue fluids and the blood within this connective tissue, whether in smaller or in larger vessels (e.g. in veins), can be displaced to other parts, provided the pressure in the former is higher than in the latter. As we have seen, the pressure in the pelvis is greater than that in the abdomen; it is therefore apparent how this intra-abdomino-pelvic pressure affects the circulation.

CHAPTER VIII

THE MEDIUM AND THE INTRA-ABDOMINAL PRESSURE: THE RETURN OF THE UTERUS

1. Displacements of the Uterus and Changes of Pressure in the Pelvis

Just as in the past it was believed the position of the uterus depends in some mysterious way on the uterus itself—that the organ stands erect in the pelvis in some strange equipoise apart from the influence of surrounding organs—so the ancients believed the uterus possesses a mobility of its own, that it is capable of wandering about the belly apart from changes occurring about it, that is, in adjacent viscera, and that especially does it do so during that peculiar state—hysteria! The round and broad ligaments of the uterus were supposed to restrain its movements; the uterus was likened to a wild beast, full of malicious and passionate desires, capable of all sorts of activities, to restrain which obviously it had to be chained by ligaments! * Such a phantasy has long been relegated to the lumber-room of recognised erroneous conceptions.

* "The round ligaments," says Astruc (1740), "are not sufficient to keep the Matrix from rolling, in convulsive Motions, to which it is very subject, and which are so strong in that Part, that Plato and Aristotle said 'The Womb was an Animal within an Animal.' In hysterick Affections, the Uterus moves sometimes upwards, sometimes downwards, with so much Force and Violence, as shews that these ligaments have but little Power over it; and we see that either a grateful or ungrateful Smell is capable of throwing it into terrible Convulsions, notwithstanding all these Ligaments can do." [5]

It is now known that in spite of the offshoots of its musculature into the surrounding connective tissue, the uterus is not capable of moving itself bodily from one position to another in the pelvis or into the abdomen; but the changes of position that occur are imparted to it, and are due to changes occurring about it; they are produced passively, not actively.

There are, as we have seen, two varieties of these physiological displacements of the uterus: the one occurring as the result of variations in volume of the surrounding viscera, and the other caused by the flow and ebb of border-line viscera in the lower abdomen, into and out of the pelvis. The former results from distension of some one or other viscus in the pelvis; and is more or less independent of change in the intensity of the visceral thrust —that is, of contraction of the abdominal wall and diaphragm—or, if this arise, it must be induced reflexly. The latter occurs apart from changes in the visceral volumes *per se*; but is due essentially to variations of the thrust from the abdomen. During movement, especially general activity of the body (e.g. locomotion), these are considerable and often great. And during visceral acts, similar variations occur. During cough, for instance, a great rise in the intensity of the visceral thrust is rapidly produced, and as rapidly passes away —appearing as a wave of pressure: during straining, the visceral thrust is also much increased, but the pressure rise is slower and is sustained. When such an act as defæcation is proceeding, with the emptying of the rectum, the uterus plainly must change in position. Under such circumstances, then, both causes of physiological displacement of the uterus are in play—change in volume of a pelvic viscus, and change in intensity of the visceral thrust.

The distending of some pelvic viscus results, as we have

seen, in very considerable changes in the relative positions of the pelvic viscera—possible because surrounding parts are so readily and easily deformable and displaceable. It affects all the pelvic viscera—at least to some extent; and since the capacity of the pelvic cavity is limited and only a small increase can occur by a descent of the pelvic floor, it at the same time causes the entry of pelvic visceral parts into the abdomen, so that the volume of the pelvic contents remains the same. This occurs from necessity, since the contents of the pelvis are incompressible—or nearly so. The intestines in Douglas' pouch are the first to be pressed upwards; but parts of the bladder and the uterus enter the abdomen when and as they become much increased in size.

So also in the other type of physiological displacement, the movement downwards of the abdominal viscera into the pelvis, or the tendency to such movement, affects the pelvic viscera collectively. It causes or tends to cause their descent—not vertically downwards in the line along which gravity acts, but downwards and backwards in the direction of the deflected visceral thrust. But although the pelvic viscera are affected collectively, they are not all affected to the same extent; as we have seen, centrally placed parts are displaced more than peripheral parts. This is especially so when the increase in visceral thrust, causing the movement, occurs suddenly; and since the capacity of the pelvis remains constant, except in so far as it is enlarged by descent of its floor, a peripheral rebound of visceral parts must occur as centrally placed parts are caused to descend—because the visceral parts are incompressible.

In this case too, then, local changes in the relative positions of the pelvic viscera are produced, similarly rendered possible by the displaceability and deformability of the visceral parts. Whilst much less conspicuous than those

caused by the first kind of physiological displacement, they have, as we have seen, much more effect ultimately—from repetition—on the relative positions of the viscera and the shape of the pelvis. And, unlike those others, with these is associated often some descent of the pelvic floor, especially when the impaction is considerable. Upon the amount of this descent, it is clear, the degree of displacement of the visceral parts will greatly depend. When it is marked and occurs easily, as when the pelvic floor is weak, not only will the descent of the centrally placed visceral parts be greater, but the peripheral ascent will be less; but when the pelvic floor is in good condition, and therefore more capable of resisting these impactions, less descent of the floor will occur, and the peripheral visceral rebound will be more conspicuous.

Both these varieties of physiological displacement are due to changes in the pressure conditions affecting the viscera in the pelvis. In both, there is a tendency to increase in volume of the pelvic contents, whilst the capacity of the pelvis tends to remain the same. But in the former, the change occurs gradually *in situ*: in the latter, it arises in the abdomen and is propagated to the pelvis. Even the small changes, the gentle rise and fall of intra-abdominal pressure produced by respiration, are said to cause changes in the position of the uterus. Much more do the variations produced by cough and general activity cause such displacements. These, we know, if excessive and too often repeated, frequently and at length, overcome the pelvic floor musculature, if this is weak or has been injured, with the production of prolapse. Similarly is the former type of uterine displacement caused by a change of pressure in the pelvis. As we have seen, the increase in volume of a viscus is associated with a rise of pressure within that viscus. It is this increase of pressure, to one or other side of the uterus, which causes the displace-

ment of the uterus ; for it occurs quite apart from increase in tone or contraction of the muscular walls of the organ in which contents are collecting. Obviously, if the pressure within the organ did not rise and become greater than that in surrounding parts, increase in its volume could not occur. Conversely, the easy distension of the viscus, which the deformability and displaceability of the uterus and other visceral parts allows, prevents the pressure within the viscus becoming greatly raised—it only becomes greatly raised when the visceral wall contracts, or the visceral thrust is increased, or distension is obstructed or rendered difficult by the pressure of surrounding parts—that is, by their becoming relatively indeformable. Thus, the pressure within the distending viscus—apart from contraction of its walls and changes in the visceral thrust—does not become or remain high, because displacement of the adjacent viscera occurs: unless the visceral walls contract, it remains approximately equal to the pressure within and between surrounding parts, which, however, as contents accumulate, progressively increases. Obviously, if a contraction of the visceral wall does occur, to the pressure due to collection of contents will be added the pressure due to such contraction ; and the pressure in the organ distending will be so much the greater than that between it and adjacent organs or than that within adjacent (and quiescent) organs.

Clearly, the passage of visceral parts from the pelvis into the abdomen, occurring with the distension, plays a part in this ; it tends to prevent the pressure in the pelvis rising. But when visceral parts cannot enter the abdomen or cannot enter it easily, as happens when a growing tumour, either of the uterus or ovary or other part, is in the pelvis and is fixed by being impacted beneath the promontory or because it is adherent to the pelvic walls—and which, besides being indeformable, is so large as to occupy

most of the pelvic cavity, i.e. is so large as to have taken the place of much of the deformable and easily displaceable tissues in the pelvis—very different conditions obtain. In such cases, when visceral parts within the pelvis distend, the pressure becomes much exaggerated. But when natural displacements can occur, and pelvic contents easily enter the abdomen, distension of a viscus within the pelvis does not cause great changes of pressure. Thus, whilst the displacement resulting from change in volume of some pelvic viscus is considerable, the change of pressure which causes it is relatively small; whilst the displacement resulting from variation of the visceral thrust —within short periods of time—is small, the change of pressure causing it is often very considerable.

The question next arises whether the visceral thrust is increased by the distending of a pelvic viscus, other things remaining the same. It is clear the passage of visceral parts from the pelvis into the abdomen, due to such process, unless compensated for, must result in an increase of volume of the visceral mass within the abdomen, however small. And it is clear, unless the abdominal wall and diaphragm relax or the posture be altered to accommodate for the increase, an increase of pressure, however slight, both in the abdomen and pelvis, will result. In pregnancy, an enormous increase in volume of the visceral mass by the growth of the ovum and expansion of the uterus and its invasion of the abdomen occurs. And in this case, when the abdomen begins to become distended, and the abdominal wall stretched, so far from a relaxation of this musculature and of the diaphragm occurring, the reverse is the case: there is an increase in their tonic contraction, and an increase of pressure is produced which persists—even during resting conditions being greater than normal.

But here a special mechanism is at work, which is not in play during the whole period of pregnancy.* During the first three months, the conditions are different; and are peculiar in that during the alternating phases of activity and rest, different nervous mechanisms become predominant. The exaggerated anteversion of the uterus, the backward and upward displacement of the cervix and the increased frequency of micturition, conspicuous during this period, are evidence that the pressure in the pelvis is greater than in the non-pregnant—an increase which is similar to but smaller than that which subsequently arises. And we must believe that during the active periods of each succeeding twenty-four hours, when the intra-abdominal pressure is already high, the result of the erect posture and bodily movements, that such an increase is present. The pelvic mass, tending to enlarge in the presence of a high intra-abdominal pressure, i.e. when the tone of the body wall muscles is good, reflexly increases (however slightly) the tone of those muscles with a consequent increase in the visceral thrust. It may do this by increasing the pressure (the exciting stimulus) on the "pressure corpuscles" Keith has described †—nervous elements about the pelvic veins in which sensory nerves end or arise, and the stimulation of which reflexly causes an increase in the tonic contraction of the body wall muscles (abdominal wall and diaphragm).

But as a result of rest, especially when the woman is lying asleep and *on her side*, different conditions obtain:

* When the pregnant uterus has become so big that it begins to stretch the abdominal wall, it directly induces an increase in the tonic contraction of this musculature by stretching its fibres.

† The existence of pressure corpuscles along the pelvic veins and the probable relationship between Paccinian corpuscles and tonus (posture tonus) of the belly wall muscles was described or referred to by Professor Keith in a series of lectures delivered at the Royal College of Surgeons some three years ago. See also [52].

the intra-abdominal pressure is now naturally diminished and such a mechanism is in abeyance. The growing ovum, expanding the uncontracted uterus, and doing so most at night because then the visceral thrust is greatly diminished, has much less effect or no effect on these pressure bodies; for the enlarging uterus naturally tends to enter the abdomen and to fall away from these sensory organs, the stimulation of which is consequently diminished. And with this, another nervous mechanism, having a contrary effect, may come into play, facilitating this movement—such, for instance, as arises when food is taken in small quantities into the stomach which has been shown to induce a lessening of the tone of the body wall muscles [64]. In such case, the increased tone of the body wall muscles present during the day, gradually gives way during the night to a marked and progressive diminution of tone, with a further fall of pressure—a fall greater than that due merely to the change in posture.

But this can only happen during the first few months of pregnancy. After the first three months, it cannot occur. The reason of this is that although the body wall muscles relax when the woman lies down, they do not do so to the same extent as earlier in pregnancy. The uterus is bigger; and now itself bulges the abdominal wall, or causes the bulging (due to other causes) to be greater. The tension of the abdominal wall not falling, its tone is not diminished. Thus, during sleep, the uterus is unable to encroach as easily as before upon the abdominal space, and the sensory bodies in the pelvis do not so readily escape stimulation. In spite of this the pressure in the recumbent position is less than in the erect posture. For this reason we may justly believe that fœtal growth is more rapid during sleep than during activity or active states. And this growth must accentuate the effect of the nervous

mechanisms in play. By increasing in volume during sleep, the uterus necessarily tends to maintain the abdominal wall on the stretch during sleep. Thus, even though the abdominal wall should tend to relax, as is likely with the protraction of the resting state (as during sleep at night), its relaxation as in the normal cannot occur. It remains stretched, therefore tense—its tonic contraction is high; at least greater than it should be. It is only before this active stretching of the abdominal wall begins, or before it has become marked, that a relaxation of the body wall muscles due to change of posture as from the erect to the recumbent, as great as in the normal, is possible. Similarly, it is only before this stage of pregnancy is reached that during the maternal sleep the growing uterus is able (by means of the other mechanism) to induce their further relaxation, whereby a further fall of pressure occurs. However this may be explained, its occurrence is indicated by the morning sickness of pregnancy, which is peculiar to this period. Certainly an extended and marked fall of pressure in the epigastrium is not without effect on the stomach and liver, and readily explains those aberrations in function of these organs in early pregnancy which result in vomiting.*

But the distending of the rectum and of the bladder seem different. Unlike the condition in pregnancy, the result of a growth and multiplication of energetic embryonic

* Sea-sickness is probably induced by similar changes of pressure in the epigastrium: it is prevented or relieved by wearing a tight abdominal belt, and much more by lying down—by either of these methods changes of pressure within the abdomen are much less readily induced by movements of the ship. Similarly, the almost uncontrollable vomiting occurring after operations on the kidney may be explained by the lateral position in which the patient is placed for the operation. In this position, in which the tone of the abdominal wall and diaphragm is always much diminished, and which is diminished still more by the anæsthesia, a marked dilatation of the stomach can easily arise.

cells, they are caused by the passage of visceral contents from some abdominal viscus into the pelvic viscus, whereby this distends—of fæcal matter from the colon directly into the rectum, or of water from the intestinal tract into the bladder, *via* the blood stream and kidneys; and this presumably will compensate for the passage upwards of pelvic visceral parts into the abdomen which the filling of the rectum or bladder causes. Thus, it would seem that the volume of the abdominal visceral mass is not appreciably altered; and so the effect this has on the tonic contraction of the abdominal wall and thoracic diaphragm does not come in for consideration, and in any case would be small. In spite of this, however, we have evidence that the pressure in the pelvis increases. Other mechanisms may come into play, such, for instance, as the increasing stimulation of the "pressure corpuscles" in the pelvis, which the filling of a pelvic viscus is likely to cause, especially when the intra-abdominal pressure is good (as in the erect posture); and this may result in an increase of the tone of these musculatures, with a resulting increase of pressure in the pelvis.*

In any case, it is clear that visceral change does induce a change in the contraction of the body wall musculatures. Thus, when the distension of a pelvic viscus has become so considerable as to induce, by stretching the

* In the endeavour to bring the pressure conditions caused by the gradual "filling" of the uterus in pregnancy and of the bladder in the non-pregnant into line, we must remember that the supply for the growth of the embryonic or fœtal cells, under normal conditions, must come from the visceral mass; whilst the supply of water for the urine at times comes largely from the subcutaneous tissue spaces. Thus, the collection of urine in the bladder may occasionally cause a real increase in volume of the visceral mass. Moreover, whilst the rate at which the fœtus grows is considerable, much greater than the growth of such tumours as fibroids and ovarian cysts, it is slow compared with the rate at which the bladder enlarges, due to secretion of urine.

visceral walls, such a contraction of its musculature as to render imminent the evacuation of the contents of the viscus, an increase in tonic contraction of the abdominal wall and diaphragm occurs—and occurs reflexly. When, for instance, the pregnant uterus greatly contracts, as at the onset of labour, a reflex contraction of these enclosing musculatures is induced—a contraction superadded to the already increased tone present in this case; and the same seems to happen when the bladder is "full." * In this way, the viscus in question is more than ever "supported"; and the act which is about to take place facilitated. This reflex increase in the tonic contraction of the abdominal wall and diaphragm, though in an exaggerated degree, also occurs pathologically; for instance, in colic, in which the visceral musculature is excessively contracted; and in peritonitis, in the early stages; and in both cases clearly is set up by sensory impulses arising from visceral parts.

And that an increase in tone of these musculatures also occurs before the content of the bladder, for instance, is maximal—that is, before its musculature is greatly contracted, when it is little contracted or not contracted—is indicated by the greater pressures found when the bladder contains urine than when it is empty, the posture being the same. But this is especially shown by the observation that artificial enlargement of the bladder causes not only an increase of pressure within

* How does a full bladder wake a person at night? My interpretation is that the bladder when full causes reflexly an increase in the tonic contraction of the body wall muscles, with a resulting increase of intra-abdominal pressure which determines an increased flow of blood to the heart, with a consequent increase in aortic pressure and a larger and quicker supply to the brain. Thus the man wakes.

In the infant the conditions are different. The bladder is a much more muscular organ, and much more independent. The brain is less developed, the blood pressure is lower, and sleep is sounder.

it, but also an increase of pressure in the rectum. Although it may be urged that in both these cases, the increase in bladder pressure may be due in part at least to an increase in the tonic contraction of its own musculature; in the second case, it is clear, the increase of pressure in the rectum can only be due to the greater pressure of surrounding parts upon it. But it has to be remembered that artificial enlargement of the bladder causes a real though small increase in the volume of the visceral mass, which must have some influence in the increments of pressure found.

The effect of variation of the bladder volume on the pressure in the rectum, I inquired into in the following way (Fig. 19). I connected one manometer by means of a flexible thick-walled tube, ending in a small rubber bag (an ordinary thin finger-stall) with the rectum, by inserting the bag partially filled with air (which the tube also contained) into the rectum. The bladder was then emptied of urine, and another manometer connected with it by similar tubing attached to a glass catheter inserted into the bladder. Two bottles, one graduated in ounces, the other filled with water, were so arranged in series and connected with the tube leading from the bladder to the manometer, that by raising the bottle containing water, the water flowed into the graduated bottle and displaced a certain amount of air contained in it into the bladder. The tube leading from this bottle to the bladder was then closed by a clip to prevent the return of air from the bladder. The bottle containing the water was then lowered until the water in it was at the same level as the water which had entered the air-bottle. The amount of air which had been displaced into the bladder was then estimated; and the pressures in the rectum and bladder were read. The patient remaining in the

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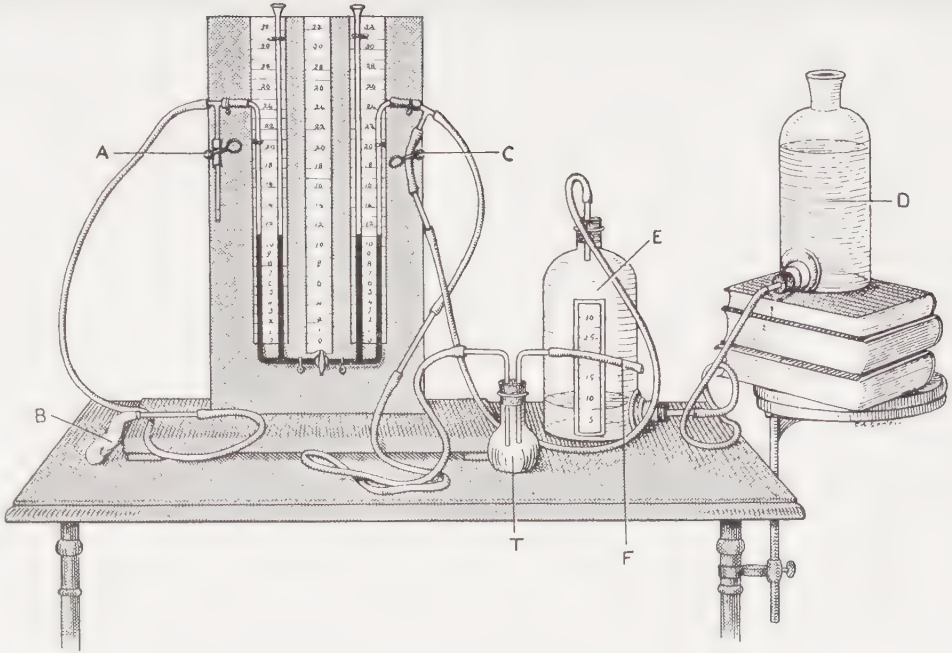

FIG. 19.—MANOMETER AND APPARATUS USED BY THE AUTHOR TO OBTAIN SIMULTANEOUS READINGS OF THE PRESSURES IN THE RECTUM AND BLADDER.

Two manometers, connected below by a tube, are fixed side by side on a board made to stand vertical. By opening the tap in the connecting tube, the four columns of mercury come level: this tap is then closed.

By means of a clip A, air can be passed into the thin-walled bag B, to distend it slightly, without affecting the pressure. The bag B is then inserted into the rectum.

The manometer on the other side is connected by a tube F (a trap bottle T intervening) with a catheter inserted into the bladder. The clip C unites this manometer with the graduated bottle E containing air; and E, in turn, is connected with another bottle D containing water. For use see text.

same position, the clip was loosened and more air passed into the bladder by raising the first bottle as before. The clip being then allowed to act, the further amount of air displaced into the bladder, and the pressures in it and in the rectum were again read. With increase of bladder pressure, which was found to occur with increase in bladder volume, and which clearly was not due to increase in bladder weight, an increase in rectal pressure also was found. By passing small quantities of air—about 5 ounces at a time—into the bladder, the experiment could be repeated three or four times in this direction. So also, on diminishing the volume of the bladder, by allowing air in the bladder to pass back to the air-bottle, easily caused by opening the clip and lowering the water-bottle, not only a diminution of pressure in the bladder, but also a diminution of the rectal pressure occurred.

In doing this, care must be taken to prevent the passage of any urine the bladder may contain into the tube leading to the manometer; for this immediately vitiates the results. This may happen either because the bladder was not quite emptied at the beginning, or, as is more probable, because urine has accumulated to some small extent during the experiment. In my first experiment this occurred. So I devised a trap in the course of this tube—consisting of a small bottle—into which any urine entering the catheter would flow and be caught. Care must also be taken that the eye in that part of the catheter within the bladder does not get occluded by coming in contact with the bladder wall; for then the changes of pressure in the bladder cannot be communicated to the manometer. It is best, therefore to have a catheter with two eyes—one on each side.

The results described are obtained not only in the left lateral position, but also when the patient is on the back and when standing erect. They are, however, more

conspicuous in the first than in the last position; an equal increment of air giving greater difference in both rectum and bladder when the visceral thrust is less than when it is greater—as was to be expected.

Let me give an example. A married woman, aged forty-nine, who had never been pregnant, came into the hospital for hysterectomy for fibroids of the uterus. The abdomen was full and the muscles "good." The girth at the hollow of the back was 31 to 32 inches; at the level of the umbilicus, 36½ to 37½ inches. Simultaneous measurement of the pressures in the rectum and bladder were taken with the following amounts of air in the bladder.

The patient lying on the Left Side:

Amount of Air in Bladder.	*Pressure in Rectum.*	*Pressure in Bladder.*
ounces.	mm. Hg.	mm. Hg.
5	6	6
10	10	10
7	8	8
4	8	6
8	8	8
10	10	11
7	8	8
With the patient standing:		
9	28	28
5	28	24
7	30	26
9	28	30
5	28	26

Here is another case. The patient (Jane Hall) was thirty-six years old, and had had seven children, the last twenty months ago. The abdomen was slightly full; and the muscles were not in good condition, as shown by some little separation of the recti and some little bulging

of the iliac regions, when the patient lying on her back raised the head. The girth at lower ribs was 31½ in. to 32 in.; at umbilicus was 34½ in. to 36 in. The rectum contained soft faeces, accounting for the relatively high rectal pressures.

On the Left Side:

Amount of Air in Bladder.	*Pressure in Rectum.*	*Pressure in Bladder.*
ounces.	mm. Hg.	mm. Hg.
5	12	4
10½	14	6
12½	15	8
9½	12	4
5	10	2
On Back with Knees flexed:		
5	20	10
10	21	10
15	22	13
19½	30	16
15	24	*18*

The higher figure obtained in the last measurement of the bladder pressure is to be rejected, for the trap-bottle was found full of urine, and when the patient coughed, no impulse was communicated to the manometer connected with the bladder. The trap was emptied and the experiment repeated—the patient still being on her back. The following figures were then obtained:

Amount of Air in Bladder.	*Pressure in Rectum.*	*Pressure in Bladder.*
ounces.	mm. Hg.	mm. Hg.
5	22	11
10	22	12
15	22	14
7½	18	10

—in which case a diminution in bladder pressure occurred with diminution in bladder volume. But the important point is, that with this diminution, a diminution in the rectal pressure also occurred, as happened in the former experiment.

The third case shows very well the result of tenesmus on the pressure in the bladder caused by distending it. The patient was old—68 years—and had come into hospital for complete procidentia for which some operation had been

On Left Side:

Amount of Air in Bladder.	*Pressure in Rectum.*	*Pressure in Bladder.*	
ounces.	mm. Hg.	mm. Hg.	
4½	6	3	
9	8	10	
13	12	13	
15½	14	19	(tenesmus) *
12	12	9	
7	8	3	
	On Back:		
7	18	11	
11	18	15	
16	20	25	(tenesmus) *
11	18	13	
6	16	10	

done. In this case, tenesmus was produced when the bladder was distended approximately to the same extent, both in the left lateral and supine positions. But, we see, the pressure was higher in the latter than in the former position. The difference was, of course, due to the difference in posture. The pressures are always higher when on the

* There was no "resting" period between this and the next reading, in which with diminution of bladder volume a fall in bladder pressure occurred. Thus we see how rapidly the effect of contraction of the bladder wall musculature on the intra-vesical pressure passed away, when the volume of the bladder was diminished.

back than when on the side; for the tone of the abdominal wall (and diaphragm) is greater in the former than in the latter position.

These cases suffice to show that the pressure in the rectum is influenced by the volume of the bladder; and that the pressure in the bladder, even when allowance is made for the effect of contraction of its own musculature, varies with its volume—due of course to the fact that other viscera are pressing upon it. It is clear, organs (in the pelvis) are susceptible to changes of pressure occurring in other adjacent pelvic organs, and that changes of pressure are caused by changes of volume of these other parts, even when the visceral thrust does not appreciably alter; just as they are caused by changes in contraction of the abdominal wall and diaphragm, whereby the pressure in the abdominal viscera is caused to vary.

Thus, the displacement of the uterus caused by enlargement of a pelvic viscus comes into line with the displacement due to variation in intensity of the visceral thrust. It is caused by an increase of pressure arising in one part of the pelvis—not suddenly, but gradually—and the displacement it causes results in an increase of pressure in the part towards which the uterus is moved. Clearly, the pressure in the tissues between the uterus and the pelvic floor is increased by the impaction caused by cough —a manometer connected with the rectum shows the rise; so also, the pressure in the rectum is increased when the uterus is displaced by the distension of the bladder. That the pressure in the bladder similarly rises when the rectum is distended and the uterus displaced forwards, I have not shown experimentally; but we can with justice assume this is so. It does so clearly in pregnancy; not only the increased pressure values found in the bladder in pregnancy, but the altered shape of the bladder – an exaggeration of the normal—clearly demonstrate it.

2. The Return of the Uterus: (*a*) from Physiological Displacements

We are now in a position to consider the peculiarity which the uterus displays when moved from its normal position: viz., that it returns or tends to return to that position when the displacing force is removed. This at any rate occurs if the displacing force, though constantly recurring, is not sufficiently powerful to permanently enlarge the pelvic cavity by causing a bulging of its walls and floor, or to force some visceral part through the pelvic floor aperture. Even in this latter case, when the exciting cause of the displacement is removed or passes away, the prolapse, whether it be of the uterus or other part, at least in the early stages of the disease, spontaneously reduces—that is to say, the parts of which it is composed return to their normal positions, or tend to do so. But when the pelvic walls and floor and the muscle guarding the aperture are not overcome, the uterus, after displacement, does return to its normal position. Such obtains in all internal displacements of the uterus, except perhaps that culminating in its retroversion; in which case the new position of the uterus is maintained by the interposition of other viscera between it and the bladder. For instance, when the distended bladder is emptied, the uterus displaced by the distension returns to its ordinary position of anteversion; and, when displaced by the impact of cough, it subsequently reascends to its original level.

What is the cause of this return in these physiological cases? The answer given in current text-books is plain. Just as it is stated the position of the uterus depends upon the integrity of its connective tissue attachments, so also, it is stated, the return of the uterus to the resting position when displaced by physiological causes, occurs

"provided the peritoneum and its reinforcing ligaments and the pelvic connective tissue possess their normal elasticity" [105]. But, it is clear, this is not the whole story; nor, indeed, is it the greater part. For just as the uterus does not change its position except in response to change of pressure about it—produced locally in the pelvis by change in volume of an adjacent organ, or transmitted to it by the abdominal viscera and caused by change in the contraction of the abdominal wall and diaphragm—so, it is clear, the uterus does not return to its original position without the simultaneous return of the viscera surrounding it, to their original resting positions or forms. In the former type of displacement, the uterus does not return without the simultaneous return of those viscera which have been displaced with it and by it, and of that which by enlarging has caused the uterus and these other viscera to be displaced. In the other case, the uterus does not return, without the return of the visceral parts above, below and about the uterus to their original positions, which is associated with the return of the parietal musculatures to their resting state or tonus, with a consequent diminution of the visceral thrust. The return in both these cases is associated with a lowering of pressure in that region about the uterus from which it was displaced: so also the return is associated with a lowering of the pressure in that region towards which the uterus was moved and which was raised by the displacement of the uterus towards it. We have indeed already seen that the displacement of the uterus causes an increase of pressure in those viscera towards which it is displaced: these in consequence suffer an increased compression, with a change in their form and displacement of their parts. Clearly then, when the increase of pressure on the one side of the uterus, which disturbed the uterus from its resting position and caused

the general pressure (in the pelvis) to rise, is replaced by a decrease, it is the pressure elsewhere—on other sides of the uterus—raised by the displacement, which determines the return of the uterus to its former position. And, it is clear, this factor plays a much greater part in the return of the uterus than the mere elasticity or recoil of its connective tissue attachments or peritoneal coverings: for the pressure conditions in the pelvis are, as we have seen, the essential causes of the uterine position.

Clearly, the return of the visceral parts, displaced with the uterus and subjected to a greater compression by its displacement, to their original positions, is due to a change in the pressure conditions: it is due to the removal of the displacing force, whereby a return to the pressure conditions, existing before the displacement, occurs—because the viscera are deformable. The cervix, which has been displaced backwards and upwards by the distending bladder, advances again when the bladder is emptied. For, even though the diminution in bladder volume may be associated with a rise of pressure within it, the result of contraction of its wall, the pressure immediately about the bladder must tend to fall as the bladder gets smaller, and it would fall unless the adjacent viscera advanced as the bladder retreated. The advance of these viscera is caused by the pressure with which they are pressed against the bladder, and which, as we have seen, is greater when the bladder is distended than when less distended—other things being equal. Not only is the bulging vertex pressed downwards into apposition with the bladder base by the direct pressure of superimposed viscera above and behind it, and by the replacement downwards and backwards between the uterus and rectum of intestinal coils displaced upwards and forwards; but readjustments of the part of the rectum behind the cervix

and of the fat-laden connective tissue about the rectum, both previously flattened against the posterior pelvic wall and so more compressed, occur in response to diminution of the bladder volume, and so of the pressure in that region. These changes, which cause the return of the viscera above and behind the uterus to their original positions or forms, together determine the anteversion of the uterine body and the advance of the cervix until an equilibrium is again established. Whilst the pressure from behind causes the cervix to advance, the pressure from above, clearly much more effective than the former, because it acts on a much larger surface, by flattening the bladder limits this movement.

Similarly, when the cervix has been pushed forwards by the distending rectum, and has caused a corresponding displacement of the bladder with a presumable increase—however slight—of the pressure within it and about it, the emptying of the rectum increases the available space in its part of the pelvis for other viscera, with a consequent tendency of the pressure in this region to fall. If the rectum were to empty almost passively, as the bladder seems to do, that is, by the pressure of other viscera upon it; or if it empties by its own peristalsis without an increase of the visceral thrust—as may happen, and perhaps often does happen *—with the resulting change in its volume and the tendency of the pressure about it to fall which the emptying of the organ causes, the cervix and posterior duplication of the bladder with adjacent superimposed parts must be pressed back into their resting positions against it, flattening it as before, by the pressure above them, which during the filling of the rectum persists or is even increased. That is to say, the return of the viscera is caused by the return of the pressure conditions to that equilibrium which caused

* Certainly under pathological conditions (e.g. in diarrhœa).

these positions to be the resting or average ones. But usually, a certain amount of straining, and often a very great deal, occurs with defæcation; and this of itself causes the visceral parts above and in front of the rectum to press upon it with a correspondingly increased force, and which indeed is transmitted beyond the rectum and causes the pelvic floor itself to descend. With the emptying of the organ, the visceral parts above it must descend; but after the act, with the opening of the glottis, the release of the diaphragm and lessening of the thrust from the abdomen, the pelvic floor reascends to its ordinary level, raising the visceral parts impinging, abutting or resting upon it, to their ordinary positions.

So also, when the cervix has been caused to descend by cough, it reascends to its resting position when the sudden increase of pressure passes away. In this, the return of the pelvic floor, depressed by the impact, plays the chief part: but the return of visceral parts situated between the cervix and the pelvic floor—the posterior vaginal wall, the rectum and the connective tissue—all flattened by the impact and their tissues spread more from side to side and so displaced laterally; and the return of peripherally situated parts, displaced upwards—both of which occur to an extent which varies inversely with the yielding and pelvic floor descent—also participate. Clearly, these visceral parts must suffer some considerable compression by the cervical descent and impaction, and some consequent deformation of their parts or displacement or tendency to such change. Thus, when the impact dies away, it leaves these tissues under a pressure greater than before, which causes their return to their original positions and forms, and thus causes the return of the cervix to its original level. In this, no doubt, the refilling of the capillaries by blood, emptied completely or partially by the impaction and so playing a

part in the displacement, is concerned in a minor way with the visceral return.

In these movements, the elasticity of the organs and of their connective tissue connections, doubtless comes into play and facilitates the return. But this is a very different conception to the notion that the elasticity is the essential cause of the return. Although the return of the uterus is associated with a change in shape of surrounding organs, both of those which have been displaced with the uterus and of that which by distending has caused the displacement, this clearly is a result, not the cause of the visceral return. It occurs in virtue of the alteration in the pressure conditions: for the (reputed) increased tension in these parts, caused by the displacement, is no more able to cause the return than their resistance to such tension was able to prevent the displacement. As the distending bladder empties, the bladder base shrinks, so that the distance from the posterior duplication to the internal urethral orifice becomes less, and with this the cervix advances. But the change in the bladder base is not the essential, the effective cause of the cervical advance; for the bladder base may be thrown into transverse folds by the greater advance of the cervix in retroversion.

Similarly, the return of the visceral connective tissue, deformed by the displacement even though it be with the production of a tension in its parts, to its resting position and a lessening of such tension, is the result, not the cause, of the visceral return. The elasticity of the connective tissue, such as it is, is small; and even though an increase be caused in it as a result of physiological displacements, the force this exerts is insignificant when compared with the effects of the pressure to which the viscera are exposed, and which, as we have seen, is a cause not only of the shapes those deformable assume—

and for this very reason a cause as well of the positions of these organs—but also a cause of the disposition of the visceral connective tissue. We have, indeed, seen that the effect of the tonic contraction of the visceral walls themselves, such as this is when the organs are quiescent and even when they contain some contents, is incomparable with the effect of the intra-abdominal pressure—it is the intra-abdominal pressure which causes the shapes of the organs, not the tonic contractions—even in the case of the non-pregnant uterus the antero-posterior flattening is probably due to this pressure. Much more, then, must the effect of the tension of the connective tissue cells and processes, of their fibrils and the strands these form, be negligible in the matter of the shapes of the viscera and of their positions in the pelvis—whether these viscera be empty or full. It is impossible to think the shape of the bladder in the adult is caused by the connective tissue attachments of the bladder, or that when the bladder returns to its empty flattened form after distension, this return or change in shape is the result of the elastic recoil of its attachments. Clearly, the exaggerated flexion of the cervix in early pregnancy, when the uterine tissues are so soft, is not the result of a tension arising in the succulent and loose connective tissue of the pelvis, which under this hypothesis must be supposed to be caused by the displacement; nor is the return of the cervix to its ordinary position after labour and the resumption of active life, the result of a recoil of its attachments.

The shapes of the hollow organs are explained by their deformability and incompressibility, whereby they adapt themselves to the capacity of the pelvis, to the positions and volumes of surrounding parts, and to the pressures obtaining. This deformability, so characteristic of the viscera surrounding the uterus, shows the small amount of tension

their walls exert on surrounding parts—we know, as a matter of fact, they are pressed against each other and surrounding parts, whatever their state of distension—so also, it shows how small must be the tension in their connective tissue attachments. These, whilst limiting wide movements, which the internal superficies of the pelvic cavity in the normal and the incompressibility of surrounding parts themselves restrict, do not limit the extent of movements occurring within the normal range of the visceral excursions. It would be strange if they did; for the physiological displacements and the return of the uterus are constantly recurring, and the tissues of the organ distending and emptying, as those of surrounding parts which undergo corresponding displacements have adapted themselves to these necessary movements. This is shown by the presence of the peritoneal invaginations, by the comparative looseness of the connective tissue between adjacent organs,* and especially of that between the viscera and the pelvic walls and floor where fat is deposited.

It may be said that, when the bladder distends, it is the upper wall, the part covered with peritoneum, which expands most, because it is separated from surrounding viscera by peritoneum; and that the base expands less, because it is adherent to the pelvic floor and walls and other viscera by connective tissue. Does not the extent of the posterior recession of the bladder in the pelvis suffice to create wonder when its limits are defined by a sound in the bladder and a finger in the vagina? How could the bladder base expand more than it does?—for even when empty it reaches back to the cervix. Only

* Halban and Tandler describe even the connective tissue between the bladder and cervix and vagina as loose.

when this and the parts behind are overcome can further recession occur. This happens when urine collects in the bladder: for the organ when full causes the cervix to be still more posterior and higher in the pelvis. It does this in spite of its base at the sides of the vagina, and its walls laterally, being connected with the pelvic floor and walls by this connective tissue. Moreover, when the pressure in the pelvis is increased, as in pregnancy, a still greater change in the relation of the bladder to the pelvic floor and walls is caused. For in pregnancy, except in the early stages, the bladder when "full" is incapable of assuming the ovoid form—the size of the pregnant uterus, in spite of the deformability of the organ when uncontracted, prevents it; and as urine collects in the bladder, whilst its base and upper wall separate, the concavo-convex form persists. Thus it is that the flattening of the bladder peculiar to the empty organ in the non-pregnant, becomes in pregnancy exaggerated. As pregnancy advances, and the intra-abdominal pressure increases, the cervix with the posterior duplication of the bladder is pressed more than ever backwards, flattening the rectum or pushing it aside, and being itself elevated. And when the limit of these encroachments is reached, as it soon is, a forward and lateral expansion of the bladder occurs, the viscus creeping upwards into the abdomen and up the lateral walls of the pelvis. As urine collects this movement of the bladder circumferentially is accentuated; as urine is voided the bladder limit recedes. These movements show the connective tissue attachments of the bladder do not serve to keep the organ in place, nor prevent its displacement over the superficies of the pelvis.

Nor can this exaggerated form of the bladder in pregnancy be associated with an increased tension of its connective tissue attachments, at least such as to prevent its further displacement. For when labour sets in, as the

child is passed downwards through the genital canal, a much greater displacement—a veritable dislocation—occurs, the bladder being bodily pushed out of the way. This happens because the child possesses volume and like the maternal tissue is incompressible, because the capacity of the pelvis is limited and the bony rim does not yield or but slightly, because the pelvic floor at first retains its position and maintains the pressure within the pelvis. Obviously, the vagina can only be dilated by the fœtus provided the incompressible tissues about it (especially the bladder) can be displaced ; for before labour, its walls are in apposition, maintained there by the pressure of these parts. And as labour sets in, these ambient parts are displaced, because with labour a new force arises within the vagina, which is propagated directly to them through its walls, and which is greater than the forces to which they are exposed from elsewhere.* Thus, as the advancing fœtal pole is pressed through the cervix into the vagina, the visceral parts about the cervix and vagina are necessarily more compressed from this part (or direction) than before ; and being deformable, they become displaced. The rectum is pressed more than ever against the pelvic wall behind, or it slips away laterally ; the bladder is crowded anteriorly against the pubic arch. And since descent of the bladder is prevented by the pelvic floor musculature—hypertrophied and in a state of good tone, and at this period of labour still occupying its normal position—and since the bladder, unlike the rectum, is not anchored inferiorly to the pelvic floor musculature except by connective tissue (!) and the elastic urethra, the bladder collectively is forced to ascend between the resisting bony pelvic rim and the distending cervix-vagina, as this last is being distended by the forced passage downwards within it

* For instance, the pressure transmitted from the abdomen at the sides of the uterus. This during the pains (the uterine contraction) is less than the intra-uterine (and so intra-cervical) pressure.

of the advancing fœtal head or other part.* Thus, we find that when the os is beginning to dilate but yet is small, the posterior duplication of the bladder reaches quite back to the cervix, far into the posterior region of the pelvis; but when the fœtal head has dilated the os and occupies the vagina, its anterior pole presenting at the vulva, the bladder is no longer a pelvic organ but has been compressed into the abdomen.

Are these displacements of the pelvic viscera in pregnancy and labour to be explained by the change in the visceral connective tissue which occurs during pregnancy? are they conditioned by it? To argue so, is to posit that the maintenance of the viscera in their usual positions in pregnancy is of an order, or of a nature, different from that obtaining in the non-pregnant—an untenable position. Clearly, if the visceral connective tissue supports or suspends the pelvic viscera in the non-pregnant, it should do so also in pregnancy. Doubtless, the disappearance of fat in the connective tissue surrounding the pelvic viscera, situated especially in that between the viscera and the pelvic walls and adjacent part of the floor, which occurs in pregnancy (Veit [102]), and which is readily explained by the increase of intra-pelvic pressure in mid and late pregnancy, plays a part in rendering facile such displacements. The consequent increase in looseness of the connective tissue, due to such loss of fat, and the increase in vascularity of all the parts, which gives to the connective tissue in pregnancy its succulent condition, help to explain the changes in position of the pelvic viscera in pregnancy; as also they help to explain the ready and comparatively sudden displacement of the bladder during

* Thus we see why straining during the first stage of labour is injurious. Straining increases the intra-abdominal pressure, the pressure of the viscera between the uterus and the abdominal walls, and thus renders more difficult the ascent of the bladder.

labour. But, clearly, the change in position of the bladder occurring with pregnancy, and the much greater one during labour—as also the changes in the connective tissue itself—are caused by pressure changes: in pregnancy, by the gradual increase in pressure occurring above and behind the bladder by the gradual growth of the uterus; in labour, by the much greater increase of pressure produced at first above and behind the bladder, next directly behind it, and finally below it, by the relatively rapid and coerced descent of the advancing fœtal part within the genital canal. Indeed, we see in the retroversion of the non-pregnant uterus—in which such changes in the connective tissue as in pregnancy have not occurred—that the connective tissue attachments between the cervix and bladder have gradually disappeared. This is not the cause of the displacement: it is the result. It is clear, the connective tissue in the non-pregnant does not suffice to maintain the uterus in its normal position; nor to prevent changes, even gross ones, from that position occurring. It may do so momentarily: but life does not consist of a moment, but of a succession of moments; and during these, as we have seen, the connective tissue, even its thickenings, adapt themselves to the position of the uterus. So also does the connective tissue in pregnancy adapt itself to the positions, and to the changes in the positions of the pelvic viscera which occur during pregnancy; the conditions in which throw light on the conditions obtaining in the non-pregnant, and confirm in an unmistakable way the opinion already expressed.

And if the connective tissue within the pelvis, in the non-pregnant, is a cause of the limitation of the movement of the bladder base, it is not easy to understand how the normal movements of the cervix take place, or how the rectum functions, the terminal part of which traverses this connective tissue. Examination shows how

the volume of this part of the rectum varies. Sometimes it is found quite empty, and its walls in apposition, so closely applied that difficulty may be presented in passing the finger beyond its folds (Houlston's valves); at other times it is found so capacious that its walls cannot be reached, as when it is ballooned. These changes are incompatible with the assumption that the connective tissue surrounding the rectum, separating it from the vagina and Douglas' pouch and from the pelvic walls and floor, undergo a tension of such magnitude that it becomes a force to be reckoned with in the maintenance of the normal positions of the pelvic viscera: that is, that they are not responsible for the visceral return.

In all these changes, it is clear, the pelvic floor, like the pelvic walls themselves, plays an important, indeed an essential part. Clearly the visceral thrust from the abdomen would project the pelvic viscera through the pelvic outlet, unless this were efficiently occluded; and the pressure in the abdomen could only be maintained, and its effect on the circulation assured, by the existence of an efficient obturator below, supporting the viscera which are compressed. The resistance of the pelvic walls and floor to the visceral downthrust and the play of all the organs during the life-history of the individual—the changes in volume they themselves undergo, and the changes in pressure caused by changes in the abdomen—are the causes of the positions and shapes of these organs. Clearly, the pelvic floor plays the essential part in causing the uterus, depressed by cough, to return to its original level, as it causes the return of other visceral parts below and about the uterus, displaced by the same movement, to their ordinary positions; for the increased compression they suffer would not arise unless the pelvic floor functioned. Nor would the pelvic viscera depressed by

straining during defæcation, reascend after the act, unless the pelvic floor also did so. So also, when the bladder empties, the return of the uterus to its normal position is conditioned by the continued resistance upwards of the pelvic floor. Similarly, during labour, the bladder would not ascend into the abdomen were the pelvic floor inefficient. Thus, occasionally, the bladder becomes prolapsed during parturition [84], which shows how erroneous is the notion that it is usually pulled up by forces from above [16].

3. The Return of the Uterus: (*b*) from some other Displacements

But the conditions we have considered are not the only ones under which the uterus when displaced returns to its original position. Even in pathological displacements under certain conditions, the uterus tends to return to its normal position. If the corsets compressing the abdomen of a pregnant woman, and which have prevented its normal distension, cease to be worn, the uterus gradually ascends more into the abdomen, increasing the bulging; and the pelvic floor previously depressed rises again, approximately to its normal level. And if, in the early stages of prolapse, the patient lies down, the prolapsed mass tends to return, and often does return into the pelvis. So also, we find when the cervix is released after having been pulled down to the vaginal orifice by forceps, it returns to its original position. In the case of the prolapse and in this experimental case, the return, like that occurring after physiological displacements, is also said to be due to the traction or tension of the ligaments on the cervix; and especially is the return in the experimental case urged as a reason for believing these attachments support or suspend the uterus in the normal, because in

it the uterus returns even when the pelvic floor is held out of the way by a speculum, and so cannot influence the movement.

It is evident, however, that the return in this experimental case and the return from physiological displacements are not comparable with each other. Clearly, the change in tension of the walls of organs physiologically displaced, as of the bladder by its distension, and of the visceral connective tissue in which all are embedded, are very much less than that caused by forcibly pulling the cervix downwards and forwards by forceps. For whilst physiological displacements are continuously recurring and easily take place, the displacement of the cervix downwards and forwards to the vulva cannot occur under normal conditions—not because the cervix is retained in place by its connective tissue attachments; but because the bladder is pressed downwards upon the pelvic floor, and being supported by it, opposes the cervical advance. This it does because the pelvic floor resists the visceral downthrust; in virtue of this, not only is the bladder maintained in position, but —because of its volume and incompressibility—it is able to maintain the cervix posterior. And it is for this reason that an undue tension does not normally arise in the connective tissue attachments of the cervix. But when the cervix is pulled down by forceps, the pelvic floor is held out of the way, and altogether different conditions obtain. Moreover, besides the traction on the connective tissue attachments of the cervix, a traction on the visceral walls themselves is produced—for instance on the bladder. Thus, whilst the tension produced in all these parts by such traction is the chief factor, causing the return of the uterus when artificially displaced downwards and forwards by forceps, this does not show that it is a tension arising in the connective tissue attachments of the cervix which

causes the return of the uterus when, for example, the full bladder is emptied; nor can it be argued from this that they support or suspend the uterus under normal conditions.

Plainly, the elevation of the uterus in pregnancy which the removal of corsets compressing the abdomen permits, is not due to the connective tissue attachments of the uterus; for the thickenings in these have all disappeared as a result of the condition. Nor does it seem reasonable to suppose that it is the tension of the ligaments of the cervix, which, when the woman lies down, causes the return of the uterus in prolapse; for, as we have seen, the ligaments have lengthened as a result of this displacement, and must therefore be much more slack when the uterus is returned to its normal position than before the prolapse originated—being indeed redundant and thrown into folds. Clearly, when the prolapse is "down," such tension as these ligaments exhibit is not comparable with that produced by dragging the normally placed cervix to the vulva; nor indeed are they the only parts involved in suspending the prolapse even in late cases.

But the reason the uterus returns in these pathological displacements becomes clear when the pressure conditions within the pelvis associated with that return are considered. For the elevation of the pregnant uterus when the abdomen is free to distend—by removal of constricting corsets—is the result of the viscera being able to readjust themselves to the altered pressure conditions now prevailing within the abdomino-pelvic cavity. Since in ordinary positions the pressure in the pelvis is always greater than that in the abdomen (although the difference must tend to become very much less when constricting corsets are worn) and in the cases of pregnancy referred

to, has been so much increased that the pelvic floor has become depressed; the return of the pelvic floor approximately to its original position explains the visceral elevation. That the pelvic floor can overcome the combined effect of the abdominal wall and diaphragm in pregnancy is not more strange than that it can successfully resist them in the non-pregnant woman. For just as in the non-pregnant it is necessary, by resisting the visceral downthrust, for the maintenance of the intra-abdominal pressure; so in pregnancy, it plays a similar rôle, for which end it becomes hypertrophied. This return of the pelvic floor to its proper level and the accompanying visceral elevation, is greatly facilitated by the assumption of the horizontal—especially the lateral—position during sleep, which diminishes the thrust from the abdomen. Thus, the change of the pressure conditions in the pelvis explains the visceral "return": that is, a movement of the uterus, not indeed to the position it previously possessed, unless it did so during sleep, for which presumably the corsets would be discarded; but to the position the uterus ought to have occupied under normal conditions.

In a similar way is explained the return of the prolapsed mass when the patient lies down; for the assumption of this position immediately lowers the pressure in the pelvis and renders easy the return of prolapsed viscera into the pelvis. For whilst the distending pressure with which the prolapsed mass is bulged into and through the aperture, dilated by this mass, is of considerable magnitude in the erect posture, and much greater during activity; when the patient lies down, this is suddenly and considerably reduced. Not only is the pressure in the pelvis lowered, but also that within the prolapsed mass. Thus, this mass not only presses less on the stretched pubo-rectalis muscle which with the pubic arch embraces

this pathologically enlarged obturator, as it does the visceral canals in the normal; but the prolapsed mass can also shrink in volume and become smaller. And since, in the early stages of the disease, the prolapsed parts form a tumour, more or less conical in shape, due to the participation of the bladder in the displacement—the apex protruding, and the base being within the pelvis, where it is continuous with the rest of the pelvic visceral mass—it is easy to understand how the pubo-rectalis muscle, in virtue of its tone, can cause the prolapse to slip back into the pelvis. It does this just as, during labour, it causes the advancing pole of the fœtal head, also conical in shape, to return into the pelvis when the pressure within the abdomen—and so in the pelvis—is diminished as the pains die away. Thus in this case, too, the pressure conditions in the pelvis, not the connective tissue, explain the visceral return.

But in many old-standing cases of prolapse, the uterus with the accompanying cystocele does not tend to return into the pelvis when the patient lies down: often it persists, prolapsed. In some of these, perhaps, this may partly be explained by the stoutness of the individual; for it is found that even in the left lateral position at ease, the pressure in the pelvis in corpulent people is still considerably high, especially if the abdominal muscles are in good condition. In other cases, the pubo-rectalis muscle may have become weaker through atrophy; but in stout individuals with good abdominal muscles and prolapse, what remains of the pubo-rectalis is generally in a very good condition. The changes which have occurred in the prolapsed mass afford a better explanation. To begin with, the prolapsed mass has become very much larger. But the chief point is that the part of the mass grasped by the muscle, instead of being conical, has become cylindrical: indeed, the

prolapsed mass is often hour-glass in shape, the distal part being greater in circumference than that compressed by the muscle. This results either from œdema of the distal part, or on account of its size—the part above, that grasped by the muscle and pressed against the pubic arch, having become thinner through continuous compression. The same occurs in hernia : the part grasped at the neck of the sac being less in circumference than distal parts. In these cases, it is easy to see why reduction does not spontaneously occur.

And when we come to examine the conditions under which the cervix is pulled down by forceps and is then released, it becomes obvious that the tension of the parts, which clearly comes into play, is not the only factor. For the position of the patient in which this experiment is performed is also horizontal—either the lateral or the dorsal, and the patient perhaps may be anæsthetised for some operation—in both of which, as we have urged, the pressure in the pelvis is low. In patients with flat abdomen, the pressure in the lateral position is 6 to 8 mm. Hg. ; in Sim's position it may be zero or negative. In the dorsal position, with the legs extended and without anæsthesia, it is higher—about 10 to 14 mm. Hg. : but the experiment could not be performed in this position. When the legs are flexed, as in the lithotomy position, the pressure is probably lower than this ; and still less during anæsthesia : but what it actually is under these conditions, I do not know. Thus, it is not surprising that, when the uterus is let go, it should return. But, obviously, the conditions of the experiment are quite different from those obtaining under natural conditions. Under these, the elasticity of the tissues, whether of the viscera themselves or of the visceral connective tissue, does not come into prominence ; and their tension is quite inadequate to cause the uterus to return.

CHAPTER IX

THE MEDIUM AND THE INTRA-ABDOMINAL PRESSURE: THE FIXATION OF ORGANS

1. The Intra-abdominal Pressure and the Fixation of Organs: the Sequence of the Visceral Connective Tissue Hypothesis

It is a natural corollary to the idea that the connective tissue thickenings of the cervix determine the position of the uterus and cause the return of the organ to its normal position after displacement, that these thickenings necessarily limit the mobility of the uterus. The thickenings of the parametrium passing from the cervix to the pelvic walls, specially postero-laterally and in an upward direction, have been thought by maintaining the cervix posterior to prevent retroversion of the organ; and, similarly, by maintaining the cervix at its level to be the essential elements in the prevention of prolapse.

We have already seen how erroneous is this conception: that the thickenings of the parametrium are secondary, not primary to the position of the uterus; that they do not prevent displacement of the uterus and under natural conditions cannot cause its return. The position of the cervix and body of the uterus alike depend on the presence of visceral parts around and about and pressing on the uterus; and on the connective tissue matrix in which all are embedded, which separates the visceral parts from the walls and floor of the pelvis and fills up all the crevices in and between them, and is subjected to the same pressure.

And just as the surrounding organs play their part in maintaining the uterus in position, not in virtue of any attachment to the uterus they may display (as of the bladder to the cervix), but in virtue of the volume they possess and their incompressibility; so the connective tissue is concerned in this mechanism, not in virtue of its thickenings connecting the cervix to the pelvic walls or of its adhesion generally to all parts, but in virtue of its mass and incompressible nature—peculiarities which it also possesses.

In a similar way do these tissues, in virtue of the conditions under which they exist, limit the mobility of the uterus. It is because the surrounding parts offer a resistance to their own deformation or displacement, that the position of the uterus is maintained; so also, it is plain, do these parts, in virtue of that resistance, limit the mobility of the uterus. But in this, the intra-abdominal pressure is essentially concerned. It is the intra-abdominal pressure which renders the surrounding parts operative. It brings into prominence as a force to be reckoned with their incompressible nature; and it does so by affecting their deformability or displaceability. For although the equipoise resulting from the interaction of the forces concerned, which gives to the uterus as to the other organs a certain position, is only disturbed (with a change in the position of the uterus) as a result of a pressure change occurring to one or other side of the uterus: yet the greater the compression of the collective pelvic contents, the greater must be the local rise of pressure causing the displacement; the lesser the general compression, the more easily will the displacement occur. The incidence of retroversion—if nothing else—shows at once this is so. In other words, the greater the pressure, the more stable the equipoise; the lesser the pressure, the less stable it becomes.

Such a relationship can only be explained by supposing that changes of pressure affect the deformability or dis-

placeability of the surrounding parts; for it is on this that the mobility of the uterus in the last resort depends. As we shall see, this supposition is justified. The resistance which surrounding parts offer to their deformation or displacement varies with the intra-abdominal pressure. The higher this pressure, the less easily are the viscera and connective tissue deformable or displaceable; the lower the pressure, the more easily can they be deformed or displaced. Thus, we see that the intra-abdominal pressure—the force arising from the compression of the abdomino-pelvic visceral mass by the combined (tonic) contractions of the enclosing musculatures (the thoracic diaphragm, the abdominal wall and the pelvic floor), a force which obviously must vary and does vary with the volume of that mass, with the development of the musculatures, and with the position of skeletal parts controlling the position of these musculatures by which they become more, or less, effective—is the essential factor in maintaining the several viscera in their respective positions; and in determining the degree of their mobility, and the ease or difficulty with which they can be displaced. And this generalisation applies to the abdominal no less than to the pelvic organs.

Such a conception necessarily is widely removed from that of those who regard the parametric thickenings of essential importance—suspending the uterus. Especially do writers of this opinion disregard the influence of surrounding visceral parts upon the position and mobility of the uterus; and deny to the intra-abdominal pressure a rôle in affecting the organ in this respect. "If a bull," writes Fothergill, "is well tethered in a field which has a small gap in its hedge, you cannot let the bull out of the field by enlarging the hole in the hedge. You must loose the bull; then he will attend to the hedge"—in which we

have suggested to us the mechanism and range (if not the cause) of the uterine mobility. The ætiological factors of prolapse are also presented. For—"Tearing the pelvic floor musculature," continues this author, "does not loosen the connective tissue attachments of the uterus, and cannot bring about prolapse. But if the parametric swing bridge is relaxed, the loose uterus comes down and gradually dilates the vaginal orifice. If this has previously been enlarged by tearing, the loose uterus descends all the quicker" [29].

In such a case, it is true, the rope tethering the bull limits the mobility of the animal: but how unlike the conditions represented in this picture is the condition of the uterus in the pelvis! Movement of the uterus is passive, not active—it is a displacement, caused by forces acting upon it, not arising within it; it depends on the movement, on the displacement and so on the displaceability of surrounding parts abutting and pressing against the organ. Clearly, we must add to the metaphor to make it more pertinent. Add haystacks—so many that the field is completely filled with bull and haystacks even to cause its hedges to bulge so that there is scarcely room to turn—and we get an image which though unlike is yet much more like the conditions to which the uterus in the pelvis is subjected. But how different then do the conditions become! In such case, it is plain, the mobility of the bull is much more restricted—to the extent indeed that the rope by which it is tethered may be almost or quite neglected as a factor limiting its mobility. And obviously, the more the bull is pressed on by surrounding haystacks, so much the more is its movement restricted —so much the more complete is its fixation.

If now we can imagine the hedge on one side of the field bulged inwards by some giant arm, sweeping haystacks and all before it, the bull—willy-nilly—will be swept in the direc-

tion of the thrust, except in so far as its rope prevents it. But what rope may that be which could stay the bull under such conditions? It is plain that the resistance to displacement of haystacks on the other side of the bull, towards which it is pressed, will be a much greater obstacle to the bull's displacement than the resistance offered by any rope; that if they are displaced, the bull must go with them—rope or no rope. If this sweeping movement is directed towards the hedge with a gap in it, the "hole"—the patent foramen!—unobstructed by any obturator, obviously becomes a source of danger through which parts pressed against it could relatively easily be pressed through—of the bull in spite of its rope, or of interposed haystacks or parts of them. So much the greater is this danger, if the hole has been enlarged by tearing.

It is, of course, true that tearing the pelvic floor musculature does not loosen the connective tissue attachments of the uterus, and cannot bring about prolapse. The injury of the pelvic floor is, as we have seen, the predisposing, not the exciting cause of prolapse. Acting alone it cannot bring about prolapse; it is only when the patient begins to get about, to cough and to strain and to perform long and arduous manual labour, that prolapse may arise; for then the exciting factor comes into play. The intra-abdominal pressure is raised as the result of the erect posture, and marked excursions due to activities are superadded to that increase. These excursions become effective in pressing out through the pelvic floor aperture enlarged by the injury, visceral parts abutting upon it, which, were the aperture normal, would be restrained. The positions of these parts—that is to say, when the uterus is anteverted, of the median basal part of the bladder and the anterior vaginal wall—being unassured, that of the uterus becomes also unstable in spite of its attachments. With their depression that of the uterus occurs, and the range of its mobility, at

least in this downward and forward direction, is increased. The parametric swing bridge, being attached to the uterus, being indeed but its vascular pedicles, is necessarily stretched by the force which depresses the uterus and so becomes longer, a mechanism readily intelligible when the magnitude of the depressing force is appreciated, and the frequency of its rises remembered. To suggest that the essential lesion in prolapse is a relaxation of the connective tissue attachments of the uterus, that this is primary and the descent or extrusion of the uterus secondary, in the presence of the increased intra-abdominal pressure and its excursions, is indeed a fictitious inversion.

It is the inevitable consequence of Fothergill's position that he is forced to declare, and without giving a vestige of a reason for it, that " increased intra-abdominal pressure cannot loosen normally fixed organs" [28]. But this statement also wants defining. For when the pressure in the abdomen and pelvis is raised and maintained raised, that is when the increased pressure is sustained, as for instance occurs when we have braced ourselves up to meet the impact of someone about to run into us, or as occurs during straining, the abdomen is much more rigid, the organs are all held in place by a greater force, and as far as their positions are concerned have become much more stable than during resting conditions or in unstrained postures. Thus we see that whilst the pregnant uterus can be moved from side to side of the abdomen when the woman lies easy, it is impossible to shift it if she is bearing down. No doubt a change in position of visceral parts occurs in the interval during which the abdomen passes from the resting or unstraining state to that of straining; but during the straining, unless some part of the enclosing muscular wall gives way, no movement occurs.

Thus, a sustained increased pressure in the abdomen

and pelvis fixes organs; it does not loosen them or move them. But this, I venture to say, is not what Fothergill means. What he would have us believe is that when the pressure in the abdomen is raised in one part, as in the epigastrium, and is propagated downwards as a wave of pressure into the pelvis and impinges on the uterus (and on other pelvic viscera), as produced by cough and manual labour, it cannot loosen the normally fixed uterus, even when the pelvic floor is inefficient, and is without effect on the parametric swing bridge. Such an opinion, as we have seen, is as unwarranted as it can be. How any gynæcologist can assert that increased intra-abdominal pressure cannot loosen normally fixed organs; and can deny its excursions to be of pre-eminent importance in the causation of prolapse, in the face of the ætiological factors so conspicuously indicating the contrary, is impossible to understand. For our part, just as we have seen that changes in the intra-pelvic pressure, whether due to the enlargement of a neighbouring organ or to changes in the visceral thrust, are the only exciting causes of change in the position of the uterus; so also have we seen that the increased intra-abdominal pressure, arising in the new-born and progressively becoming greater with growth and increasing activities, is the only exciting cause of the ontogenetic displacement of the organ, just as—it is plain—that that pressure in the adult, whether generally raised (as by tumour formation) or whether temporarily raised frequently (as by cough) is the only exciting cause of the pathological displacements of the uterus.

Unhappily, the declaration that increased intra-abdominal pressure cannot loosen normally fixed organs, leads to the further declaration that normal organs are fixed by their connective tissue attachments. The uterus, vagina and bladder, says Fothergill, are anchored or fastened by the firm, dense connective tissue which forms sheaths for the

arteries, veins, lymphatics and nerves which supply these organs [28]. It is these which keep the viscera in place. And the proof, he thinks, is plain. For were the organs loose; were they "held in position only by their shape and by the so-called ligaments" (i.e. by the round, the broad, and the utero-sacral) "they would be expelled"—so we read—"with every motion of the bowels and would hang outside the body as in cases of complete 'prolapse'" (Fothergill [28]). But this statement, suggesting that during straining the pelvic viscera are retained within the pelvis by their connective tissue attachments, is also unwarranted. It is, however, so plausible that at first sight it seems it might be true. It is indeed so plausible that it has been accepted without examination. But before we examine it, let us consider first the other statement—co-ordinate with it—which this plausible idea is supposed to confirm.

2. The Variations of the Uterine Mobility and of the Pressure in the Pelvis

The statement that normal organs are fixed or fastened by their connective tissue attachments to the pelvic walls, whereby their several positions are assured, is, as a matter of fact, far from the truth. In the pelvis, when the intra-abdominal pressure is not greatly raised, all the organs are mobile: none are fixed—none are so fastened by their connective tissue attachments that their mobility under natural conditions is restricted. Only in the region of the pelvic floor aperture, near their terminations, are the rectum and vagina fixed—elsewhere, their walls are freely movable. Not only can the passages be dilated, but they can be bodily shifted. Post-rectal swellings displace the rectum; similarly, the vagina is displaced by tumours behind it—e.g. in Douglas' pouch, as in ordinary retroversion and in a more marked degree when the retroverted uterus is pregnant. The same applies to

the bladder. Not only can its walls separate as urine accumulates; but the changes in form in pregnancy and the bodily displacement during parturition which occur, show the organ is not fixed to the pelvic floor or walls.

Similarly, the uterus, in the normal and under resting conditions, is mobile. On bimanual examination, the uterus can be moved upwards and downwards, forwards and backwards, and from this side to that. Immobility of the non-pregnant uterus—its fixation—is pathological: it shows something is wrong. Either some tumour is present in the pelvis, and is sufficiently large to prevent or to greatly limit movement of the uterus; or fixation has occurred as the result of inflammatory disease. Inflammation of the connective tissue in the pelvis is especially associated with such fixation. So also the presence of scar tissue, the result of the healing of wounds by granulation tissue, for example of tears of the cervix extending into the bases of the broad ligaments caused by labour, limits the mobility of the uterus. But under natural conditions—that is, in health—the uterus in the majority of cases is easily mobile. Many writers have referred to this feature. "The normal uterus," say Winter and Ruge, [105] "is movable within wide limits." But its condition of mobility is, I think, best summed up by describing the uterus as an organ floating in the pelvis; for this suggests the ease with which it may be caused to move, both by the finger on examination, as also by the natural variations in volume of surrounding parts. Matthews Duncan, more than any other author I have come across, dwells on this: "The uterus," he says, "is often justly described as to some extent a floating organ" [24].

But when we come to examine many different women, we find there is a good deal of difference in the mobility of the uterus—that is, even in the normal: in some,

the uterus is much more mobile than in others, even apart from pathological conditions, and when the woman is at ease. Thus, considerable distension as of the bladder, or of the uterus itself in pregnancy, markedly diminishes the mobility of the uterus. And more general conditions affect its mobility. The uterus can be moved bimanually in many cases, much more easily and to a much greater extent in thin than in corpulent women; and in patients with weak abdominal walls than in those in whom they are well developed. Especially when these conditions are combined do great differences obtain: in fat and strong women the uterus—bimanually—is much less mobile, even though inflammatory disease can be excluded, and in the absence of tumour formation.

What is the cause of this difference in the mobility in these respective cases? According to the connective tissue hypothesis, the difference should depend on a difference in the connective tissue thickenings or of the perivascular connective tissue in the different cases: according to our opinion, it should depend on other causes. It should depend on a difference in the intra-abdominal pressure, which transmitted to the pelvis, causes the organs in the pelvis to be more easily or less easily deformable or displaceable. Clearly, when the uterus is easily mobile, the visceral parts about the uterus (viscera and mesodermal matrix) are correspondingly easily deformable or displaceable. But if these parts become less deformable or are replaced by indeformable tissues, however loose be the connective tissue thickenings—the pedicles of the organs—the mobility of the uterus will be restricted, or the uterus will become altogether immobile. This must necessarily happen, because the pelvic cavity is not elastic, and the visceral parts are incompressible.

The latter is the valid conception. We have evidence

that the deformability or displaceability of these parts about the uterus does vary with their compression, and that with this the mobility of the uterus varies. The mobility of the uterus is different when the pressure conditions obtaining are different—both in the same individual at different times, and in different women at the same time. We find that when the pressure is low or is constantly low, the parts are easily deformable and displaceable; but when the pressure becomes raised or is constantly high, they are less deformable: that is to say, with such pressure differences, the mobility of the uterus varies. This applies to local conditions of pressure occurring in, and being more or less restricted to, the pelvis; as also to conditions affecting the whole abdomino-pelvic cavity.

The bladder is less deformable when distended than when empty; because not only is the pressure within it higher in the former than in the latter state, but the pressure in other parts of the pelvis is also raised, for instance, in the rectum; and the diminished mobility of the uterus is plain—greater increments of pressure on one or other side of it are necessary to move it than before. So also, in early pregnancy, the uterus is less mobile than in the non-pregnant state. The increase in its volume, associated as it is with an apparently increased anteversion and with changes in the position of the bladder and cervix, must be associated with an increase of pressure in surrounding parts. Such pressure in the pelvis, when viscera within it are enlarging, and can easily cause the displacement either of other pelvic organs (e.g. intestines) or of parts of themselves, into the abdomen, is clearly associated, as we have seen, with a continuance—even an increase—of the visceral thrust.

The restriction of the uterine mobility with the appearance of a growing tumour in the pelvis, results from the

same cause—a gradually progressive increase of pressure in the pelvis. But whereas the uterus when pregnant, and the bladder in all cases, increase in volume relatively quickly, and can therefore easily cause reflex nervous mechanisms to arise with an increase of pressure; the growth of tumours is relatively slow and consequently affects less readily the nervous machinery, and the rise of pressure resulting is less marked. And whilst the increased compression of the viscera renders a pregnant uterus or a bladder partially distended, less deformable, even when the muscular wall of the organ is uncontracted and the other tissues remain unstretched; tumours consist of indeformable tissues from the start, i.e. even apart from such compression. In the case of tumours, such as ovarian cysts and fibroids of the uterus, the indeformability is due to the pressure within them, and not to the pressure outside them, between them and surrounding parts. Cysts become flaccid only when punctured or burst; similarly, when the capsule of a fresh and unhardened fibroid is incised, it retracts, and the parts inside tend to extrude—as also happens in the case of the normal non-pregnant uterus itself. Thus, tumours affect the mobility of the uterus by encroaching on the space in the pelvis occupied by the normal tissues, and replacing easily deformable by less deformable or by indeformable tissues.

But tumours also cause the pressure in the pelvis to rise, as the figures given by Hörmann (in spite of his contrary opinion) incontrovertibly demonstrate [44]. Thus, in the case of a fibroid uterus reaching to the navel, the pressure in the rectum in the erect posture was 22 cm. of water, but after removal a rectal pressure of 16 cm. of water was found for the same posture. Similarly, in the case of a large ovarian cyst, the rectal pressure before operation was 26 cm. of water, but after 19 cm., the

posture in each case being the same. Thus, besides the replacement of deformable by indeformable tissues, an increase of pressure is produced, whereby the uterine mobility is further restricted. But clearly, the mobility of the uterus can only become markedly restricted when the space of the pelvic cavity occupied by such material becomes great—unless the pelvic pressure is also considerably raised. A small ovarian cyst does not prevent movement of the uterus: it is only when the cyst becomes large that it has this effect, and especially when it is unable to enter the abdomen does the uterus become relatively fixed; and this, as we have seen, is associated with a very great increase of pressure in the pelvis. So also, in inflammatory disease in the pelvis, the mobility of the uterus is much reduced. In this case, too, we have a transmutation of the easily deformable connective tissue into indeformable masses, within which, too, the pressure is considerably raised—since, on incision, an exudation escapes. But, besides, the general compression is increased by the reflex increase in tone of the belly wall muscles.

These are instances in which the increase of the average pressure restricting the mobility of the uterus arises in the pelvis. But the same happens when the average pressure within the abdomen becomes increased—the increased visceral thrust, of course, influencing the pressure in the pelvis: this also causes a diminution of the uterine mobility. For instance, the diminished mobility of the uterus in mid- and late pregnancy, which occurs in spite of the easy deformability of the organ when uncontracted, is explained by the rise of pressure in the abdomino-pelvic cavity which the rapid increase in volume of the uterus causes. And the same occurs in the non-pregnant and apart from tumours: when the average pressure is good

the uterus is less mobile than when the average pressure is not so good—as extremes, the diminished uterine mobility in robust corpulent people, and the increased mobility in patients suffering from enteroptosis, may be cited.

At first sight, it would appear that the size itself of the uterus in pregnancy plays a part in its diminished mobility; and so it undoubtedly does when it is indeformable, i.e. when it is contracted; but when uncontracted, it is so soft and deformable that it can have little or no effect in this respect, except in so far as the fœtus within it limits its deformability. The difference found in the mobility of the uterus in primægravidæ and multigravidæ respectively, shows that apart from mere size the mobility is greatly affected by the intra-abdominal pressure—that is, by the degree of tone manifested by the abdominal wall and diaphragm. In the later months of pregnancy, as in the seventh or eighth, the body of the uterus can be shifted much more easily from side to side of the abdomen in a woman who has already borne children, than in one pregnant for the first time. And whilst in primægravidæ, the lower uterine segment, distended by some part of the fœtus, occupies the pelvis and is pressed low down, quite near to the pelvic floor; in multigravidæ the lower uterine segment is generally at a higher level, not infrequently above the brim, in which case the fœtal part within it, e.g. the fœtal head, is freely movable, as from one iliac fossa to the other. With these differences, the abdominal wall is found to bulge less and to be more tense in the former than in the latter, which condition can only be due to a difference in the tonic contraction of the abdominal wall musculature in the two cases. So also, the diaphragm in primægravidæ is thrust more upwards into the chest than it is in multigravidæ, as is shown by the position of the heart in these respective cases [95]; and this, which causes an increased stretching

of the diaphragm, is associated as in other cases with an increase in its tone [76]—i.e. with a greater pressure downwards on the epigastric viscera.

But in non-pregnant women, and in the absence of tumour formation, such a cause (increase in volume of the uterus) of change in the uterine mobility does not obtain; and the effect of the pressure conditions within the abdomen is rendered more conspicuous. In corpulency, as in pregnancy, the increase of pressure within the abdomen, and so in the pelvis, is associated with an increase in mass of the abdomino-pelvic contents, due in the main to an increase in the deposit of fat in the visceral connective tissue—in the omentum, mesenteries, renal regions, subperitoneal tissues, and in the pelvis about the rectum, uterus, vagina and bladder; though the increase in contents of the digestive organs, and the increase in mass of the viscera, necessary to deal with the increased intake of food, at least with the raised metabolism, also participate. And, as in pregnancy, the pressure increase is caused by an increased tone of all the enclosing muscles which in robust corpulent people have become hypertrophied.

That such increased compression of the viscera (even during resting conditions) is present in these people is shown immediately by the pressures found in the pelvic organs, and is explained by the greater tone exhibited by the body-wall muscles when hypertrophied. In a patient I examined, nineteen years of age and eight to ten weeks pregnant, and who was exceedingly stout (the girth at the umbilicus measuring 37 inches), the pressure in the rectum in the left lateral position, at ease, was 18 to 22 mm. Hg.—a pressure as great as that found in well-developed women with flat abdomen in the erect posture. And that the uterus is less mobile in such people, even when non-pregnant and although the contents of the pelvis be normal, than in healthy women with flat abdomen, is apparent on

bimanual examination. Not only is the uterus more difficult to move, but it is also more difficult to define. This, no doubt, is due, at least in part, to the thickness of fat in the subcutaneous tissues and to the tone of the abdominal wall ; but it is also due to the increased compression of the visceral parts about the uterus, which has rendered them less deformable and which therefore, besides restricting the mobility of the uterus, masks to greater extent than in other cases the limits of the organ. How, then, have these visceral parts become less deformable ?

3. The Intra-Abdominal Pressure and the Deformability of the Medium

In discussing in a previous chapter the deformability of the visceral parts about the uterus, we saw this depends, (1) on the deformability or elasticity of the tissue cells, either singly or collectively, of which these parts are composed ; (2) on the presence of cell-spaces and intercellular channels permeating the tissues, containing liquids more or less easily displaceable ; and (3) on the existence of invaginations of the peritoneal sac lining clefts between viscera, and on the separation of the walls of the hollow organs by the collection of contents more or less mobile. Each one of these factors may become predominant in affecting the mobility of the viscera. Contraction of the non-striped muscle cells renders the viscera—not only the uterus—indeformable : turgidity of the capillaries and distension of the cell spaces (as in inflammation) converts soft parts into hard : and marked distension of the organs themselves, even when their muscular walls are uncontracted, renders them less deformable or even indeformable. In healthy corpulent people leading active lives, which of these factors have varied, rendering the viscera less easily deformable ?

We may immediately rule out of court the influence of the peritoneum and of the contents of organs: these remain the same and affect the viscera as before. And we may dismiss an increased contraction of the non-striped musculature of the organs, such as the autonomic nervous system may produce. We have indeed seen reason to believe that in the presence of a persistently raised intra-abdominal pressure, the muscular walls of the hollow organs are even less contracted than when the visceral compression is less. We cannot believe that when the bladder is more compressed, as it is in robust corpulent people, and the pressure within it is therefore greater than in women with flat abdomen, the bladder wall is more contracted in the former than it is in the latter, other things being equal, or indeed that it is contracted at all; for this would still further raise the intra-bladder pressure and entail a correspondingly greater work on the kidneys and ureters in secreting and excreting the urine. If the non-striped muscle wall of the quiescent organ exhibits a "tone," this must result from the metabolism of its non-striped muscle cells (and of its other cells) rather than be due to nervous impulses.

And the visceral parts about the uterus do not consist only of non-striped muscle cells, but of collections of epithelial cells and of other (mesoblastic) cells: in robust corpulent people all these have become less deformable, more elastic, the tone of all has become increased. But besides this change, a change in the cell-spaces and in the capillaries has occurred whereby their contents have become less easily displaceable than in the non-corpulent state; and thus movement of cell upon cell (apart from their deformation) is rendered more difficult. In corpulency, the conspicuous change is in the cells of the connective tissue, in which the pelvic organs with the invaginations of the peritoneal sac are embedded, which separates organs

partly from each other and all from the pelvic walls and floor, except in the region of the aperture.

These have become laden with fat: their volume has become greater, and the tension within them much increased. Because of this, they press on each other with greater force, and obviously are less easily deformable. For the same reason, the cell-spaces between them and the capillaries permeating the tissue they form are more compressed. Not only are these spaces and channels more compressed because the fat cells themselves press more closely together, but much more because the general visceral compression is raised. And the more lymph the cell spaces contain, and the greater the volume of the blood in the capillaries (since these must be contained under a greater pressure than obtains in the non-corpulent state), the more difficult is the movement of individual cells on each other and the more indeformable is the tissue which these cells, cell-spaces and capillaries comprise. The changes in these two factors which react on each other, of which those in the latter plainly depend on the raised intra-abdominal pressure, together result in an increased firmness of this connective tissue, and thus the viscera embedded within it are less easily displaceable.

But though the conspicuous change is in the connective tissue, we can hardly believe that other tissue cells, the essential cells of organs, remain unaffected. For the changes which produce an increased metabolism in connective tissue cells (whereby they become laden with fat), affect also these other cells and their metabolism is also raised; from which cause they can more readily maintain the spherical form or the form they have inherited, in the presence of environmental forces of the same magnitude as those acting before —i.e. when the individual possessed a flat abdomen, and the intra-abdominal pressure was not so high. In robust corpulent people, then, not only are the viscera packed

more tightly together ; but besides this, the visceral parts about the uterus are less deformable. Thus, opposing parts give way less readily ; and visceral surfaces cannot glide so easily over each other, either when these surfaces are large, as in the case of the peritoneal sac, or smaller as in the case of opposing mucous areas, or quite small as in the cell spaces. Thus greater changes of pressure must occur to cause the same visceral movements or displacements as before, and in consequence the uterus is less mobile.

Whilst the diminished mobility of the uterus in the case of robust corpulent people is perhaps more easy to understand because of the nature of the case, we see the same occurs when an increase in volume of the visceral mass is produced in other ways. The distending of the bladder, the enlargement of the pregnant uterus, the growth of tumours in the pelvis, all lead to a diminution in the mobility of the viscera. But, as we have noticed, in each of these cases, the increase in volume of the visceral mass is not the only factor : an increase of the intra-abdomino-pelvic pressure also occurs. This is common to all these cases which differ in other respects, and plainly is the determining cause of the lessened mobility. Thus, these cases come into line with those in which the relative or absolute fixation of the organs is produced, as for instance on straining ; when instead of such increase in volume of the visceral mass, that volume remains the same, but the pressure is increased by an increase in the contraction of the enclosing musculatures.

Such increase of intra-abdominal pressure, of brief duration and much greater extent, and thus differing in these respects from the increase referred to in robust corpulent people, by affecting the visceral blood stream, compressing the large and extensive venous channels and the enormous

visceral capillary bed, must clearly diminish or even temporarily abolish the deformability of the visceral mass. The pressure in the abdominal and pelvic veins rises greatly, for the pressure in the thorax is greatly raised: the glottis is closed and a considerable expiratory effort made which for the time being prevents or checks the passage of blood from the abdomen. It impedes the blood-flow through the lungs and compresses the heart, influencing the distension of the auricles and great veins leading to them. Thus, the superficial veins swell (e.g. of the forehead), causing an engorgement of capillaries (e.g. of the face). The same happens in the abdomen. The abdominal veins distend, and the blood-flow through the visceral capillary beds is checked with their temporary distension. Since the intra-abdominal pressure at the same time is greatly raised, the same effect on the deformability of the visceral mass is produced as occurs in lesser degree with enlargement of the visceral mass associated with an increased tone of the enclosing musculatures, when the bladder distends, or the uterus enlarges, or fat is deposited.

That the increased intra-abdominal pressure, or the increased visceral compression, plays the predominant part causing this diminished mobility, besides being shown by its effect on the visceral capillary blood-flow, is also shown by the influence it has in preventing retroversion of the uterus. As we have seen, the uterus only becomes retroverted when the intestines behind the uterus can pass easily upwards and forwards into the abdomen, and this obviously is less easy when the intra-abdominal pressure is high than when it is low. It cannot occur when the intra-abdominal pressure is raised, as when we brace ourselves up to meet the impact of impinging bodies, or during straining.

Thus is explained the influence of trauma in producing retroversion. When shocks are received unexpectedly, i.e. when the intra-abdominal pressure is not raised to meet environmental forces, displacements of visceral parts can clearly much more easily occur, than under other conditions. When a fall occurs, if the bladder be partially distended and the uterus be more or less vertical in the pelvis, it is easily intelligible that the sudden impact caused by reaching the ground should cause the uterus to swing backwards and displace upwards intestines behind. For during falling, the intra-abdominal pressure probably is reduced—one notices the sinking feeling experienced at the beginning of a descent in a lift (i.e. during the period of acceleration from rest to the rate of descent subsequently occurring). During an unexpected and ungoverned descent, in which a condition of statics is replaced by a condition of astatics, the nervous machinery controlling the intra-abdominal pressure is probably thrown out of gear; with a probable reduction of pressure and resulting instability as regards their relative position of visceral parts behind, or above and behind the uterus, in virtue of which the uterine position becomes unstable. And the thinner the individual, the more likely is this to occur.

In the case of robust corpulent women, under ordinary conditions of life, the effect of the increased intra-abdominal pressure is plainly before us. The peripheral ascent of visceral parts due to visceral impactions from above occurs less readily in these cases because of the increased compression and increased viscosity of the visceral mass. These parts, by being maintained in position, even when visceral impactions occur, maintain the body of the uterus anteverted and prevent the entry of intestines between the uterine fundus and bladder. Nor can retroversion be easily induced, or indeed at all

induced, in robust corpulent people. This is so, not because of the increase in mass of the visceral connective tissue *per se*—the deposit of fat—but because of the pressure with which all the visceral parts are pressed together, and which besides compressing them to a much greater extent, has rendered them less easily deformable and so less easily displaceable. The effect of the increased indeformability or viscosity of the fat-laden connective tissue in which the cervix is embedded on the cervix is obvious; but the anteverted body of the uterus is similarly affected by the lessened deformability and displaceability of the parts around it, due to the greater metabolism and so greater tone of all the cells of which these parts are formed and the greater pressure to which they are subjected.

But in thin and ill-nourished patients, retroversion can often be bimanually caused. Here the fat in the visceral connective tissue has disappeared, or nearly so, and the visceral connective tissue is much more loose. So also the viscera themselves, except in so far as their muscular walls may be contracted, possess much less tone, or are in a toneless state. Thus it is not surprising the uterus should be easily mobile; not only that the cervix can be easily displaced, but also that the fundus can be retroverted bimanually. But the atrophy of the visceral connective tissue, whilst explaining the increased mobility of the uterus, is not its cause; which is due to the lower level at which the intra-abdominal pressure is maintained—in these cases the average pressure being much less than in corpulent people.

That this is so is shown by the condition in pregnancy. For as pregnancy advances the fat in the pelvis also disappears, the cells containing the fat become smaller and shrink, so that the spaces between them become relatively larger. This, coupled with the increased vascu-

larity of the pelvic tissues in pregnancy, gives to the visceral connective tissue its succulent nature in this condition. Thus, the visceral connective tissue at least becomes less viscous in pregnancy, even than it is in well-developed women with flat abdomen; but, in spite of this, the mobility of the uterus and other viscera is diminished. This is explained, as we have seen, by the rise of the intra-abdominal pressure, caused by the rapid rate of growth of the uterus and the effect this has on the abdominal wall and thoracic diaphragm, whereby their tonic contraction is increased. It is plain, therefore, that in thin, ill-nourished women, the increased mobility of the uterus, whilst being allowed by the decrease in mass of the parts about the uterus, is due to the diminished intra-abdominal pressure obtaining in such cases. In such, the displacement of intestines behind the uterus upwards into the abdomen presents less difficulty, so that retroversion can more easily occur.

In enteroptosis, the uterus is still more mobile. When the tonic contraction of the abdominal wall and diaphragm is maintained continuously reduced, as it is in this condition, all the viscera are easily mobile, and especially the uterus. That the intra-abdominal pressure is deficient in these cases, is shown by the position the abdominal wall assumes when the patient stands up. Patients thought to be suffering from this disease should always be examined in the erect posture: the bulging of the lower abdominal wall becomes plainly evident in this position, and is associated with a recession of the upper part, a drooping of the shoulders, an increased obliquity of the ribs, and a lessened antero-posterior diameter of the chest.

Whether in robust corpulent people the thickenings of the visceral connective tissue in the pelvis are more

prominent, whether they are tougher and more gristle-like in structure and possess greater strength than they are said to be and possess in well-developed non-corpulent people (e.g. by Tweedy [100]), I am not in a position to say. Since, however, in the former type of individual, the pelvic visceral tissues are less deformable and are constantly more compressed, from which causes visceral displacement (that occurring naturally as that produced on examination) is more difficult than in the latter type, it would seem to be otherwise. It would seem that the connective tissue thickenings which are thought by so many to support the uterus and limit its movement and cause its return after displacement, do not hypertrophy, even when the intra-abdominal pressure—the pressure tending to thrust the pelvic viscera downwards through the pelvic outlet—is maintained greatly increased. Certainly, if these thickenings are well developed at all, it must be in the thin and in the weak ; in women in whom visceral excursions are marked because of the disparity of the rises of intra-abdominal pressure, the resultant of activity and cough, from those pressures obtaining during resting or static states.

But whether the visceral connective tissue attachments of the uterus—the uterine pedicles!—hypertrophy in corpulency or not, does not greatly matter, as a comparison with the conditions in pregnancy will convince one. Here, although the pelvic floor hypertrophies as it does in corpulency, the thickenings of the visceral connective tissue disappear, so that the reason assigned to them in the non-pregnant for supporting the uterus and limiting its mobility—that they offer considerable obstacles for hysterectomy—is no longer applicable : and this fact confirms and emphasises perhaps more than any other our general contention—that it is the walls and floor of the pelvis and not the visceral connective tissue which

support the pelvic viscera and oppose the visceral thrust. It confirms in an unmistakable manner the opinion that the mobility of the uterus is dependent on the intra-abdomino-pelvic pressure, and is conditioned by the far-reaching effects of the same physical force.

CHAPTER X

THE PELVIC FLOOR AND THE VISCERAL MASS IN THE NORMAL DURING STRAINING

1. The Pelvic Floor during Straining: Defæcation and the Reason of Straining

In the absence of such a conception, the idea that the connective tissue attachments suspend the pelvic viscera during straining, preventing extrusion, is plausible enough. Indeed, under such conditions, it seems the only explanation of the visceral retention. For on straining the pelvic floor descends and the pelvic floor aperture is dilated, so that even if the pubo-rectalis embraces the visceral canals as an obdurator under other conditions, it can be stated with perfect truth that it does not do so—at least to the same extent—during straining; that is, during a state in which the pelvic pressure is considerably or greatly raised. And straining is not infrequent. Although peristalsis is essential for efficient defæcation, straining is often associated with the act, often preceding it, and indeed often occurring without it, as when defæcation is attempted in its absence, and even in these in the vast majority of cases prolapse does not result. How then are the viscera retained during straining?

It is, however, clear that the size of the aperture and the condition of the muscle forming its postero-lateral boundaries are of very great importance in the prevention of prolapse during straining. One cannot believe that prolapse

can occur as it so often does apart from straining (as for instance from cough), in which cases as all the world knows the injury of childbirth plays such a predominant part as a predisposing cause, and yet think the size of the aperture during straining is without effect on the retention of the visceral parts during that act. And in the next place, it is necessary to notice that the pelvic floor aperture is not much enlarged by straining. In the absence of extrusion of bodies from the pelvis, either from within the visceral canals, or of visceral parts themselves, which can be insinuated in a wedge-shaped sort of way into the pelvic floor aperture and so dilate it, this aperture is but little increased. This increase is mainly antero-posteriorly; and on measurement—i.e. under experimental conditions, the individual lying on her side—is found to be in the normal about half an inch [73]. But from observation on one's self, one knows that though the pelvic floor descends on straining, and that its superficies in the antero-posterior plane is increased, the descent of the floor even when the crouching attitude is assumed is surprisingly small. In this descent, the pubo-coccygei (because of their attachments), are most affected, and the part of the aperture they bound is most increased antero-posteriorly; but the pubo-rectalis is less affected. An enlargement also takes place transversely; but what this is, it is difficult to say. It would seem that, owing to the position of the aperture in the anterior part of the pelvic floor, within the pubic arch, and to the fact that the floor descends most more posteriorly, between the ischial spines, the transverse enlargement of the aperture is certainly not more, and probably less, than the increase antero-posteriorly.

And, in the third place, it is necessary to remember that during straining the pelvic floor musculature still functions. Although depressed, it is still contracted: indeed, since the intra-abdominal pressure is much increased, the contraction

of the pelvic floor is correspondingly increased to successfully oppose it. That is why the aperture is so little enlarged by straining. The pelvic floor is depressed by the combined contractions of the thoracic diaphragm and abdominal wall, because the attitude of the body assumed is such that a maximal pressure can be brought to bear on it ; but its depression is small and is obviously limited by its own resistance.* According to Halban and Tandler, palpation of the muscle during the process of straining shows that as the floor is depressed its contraction is increased. But the retention of the viscera within the pelvis during straining also shows this. We cannot believe that such an extensive organ as the bladder is retained within the pelvis during straining by its connective tissue attachments ; and that these prevent the organ bulging the pelvic floor downwards to a greater extent than normally occurs, or that they prevent that part of the bladder related to the pelvic floor aperture from being extruded through the aperture whilst this is enlarged by the straining. Several reasons convince us of this. To begin with, the thickenings of the connective tissue in which the bladder is embedded are so insignificant that it cannot be supposed they play any rôle in the maintenance of the position of the organ whether the woman be straining or not. Next, we know that during the first stage of labour, when the bladder is still a pelvic organ, its attachments are so loose that as labour proceeds the bladder suffers such an extreme displacement that it becomes again an abdominal organ. If the connective tissue attachments of the bladder prevent extrusion of the organ during the straining which so often occurs during the first stage of labour, they ought to suffice to prevent its bodily displacement into the abdomen in labour. Yet the bladder is retained in the pelvis at such times by

* Even in marked straining (apart from parturition) the pelvic floor only descends about half an inch [73].

something in spite of the dilatation of the pelvic floor aperture; for occasionally during labour cystocele occurs. But we cannot believe that in these cases its extrusion results from a failure of its connective tissue attachments.

Is it then to the vagina that such maintenance is to be imputed? Does the anterior vaginal wall which passes obliquely upwards and backwards from the pubic arch to the cervix, beneath the median basal part of the bladder, play any part in sustaining this part of the bladder in place during straining? It might be supposed to have some such effect, because of its continuity with the cervix and the attachment of this to the pelvic walls by the parametric swing bridge or by the utero-sacral ligaments —it might be supposed to become taut during the descent occasioned by the straining. But these attachments of the cervix even in the non-pregnant are such that they suffice to allow a greater movement than that caused by straining, which renders impossible such tautness of the vagina arising. Moreover, during the first stage of labour, when straining occurs, one can often see the pelvic floor descend and on examination find this caused by the fœtal head enveloped by the undilated cervix. But then one finds the vagina, instead of being taut antero-posteriorly, all thrown into transverse folds. Clearly, the supposed rôle of the thickenings of the cervical connective tissue in such a mechanism is out of the question. How can the scanty and loose connective tissue in the pelvis during pregnancy suffice to prevent extrusion of the organs during straining? The reason the bladder is retained in the pelvis during straining in the pregnant as in the non-pregnant woman is because during the straining the pelvic floor still functions, and in virtue of its resistance to the downthrust maintains the viscera in their now lowered positions.

It is indeed because the pelvic floor is such an efficient mechanism occluding the pelvic outlet, and so successfully opposes the tendency to visceral descent and extrusion, that straining is so often necessary for visceral acts. For these to occur, the pelvic floor aperture must usually be dilated—to a degree corresponding to the calibre of the body to be passed; and the pelvic floor correspondingly depressed. But the pelvic floor resists these changes: and it is this which causes the straining to arise or to be greater than it otherwise would be, and which renders visceral acts often so difficult. In other words, the musculature guarding the aperture obstructs the way through; and in order that extrusion may occur, and the aperture be sufficiently dilated, the musculature has to be overcome.

In this, the body to be passed plays the essential part. Normally, it is propelled by the viscus within which it is contained towards the pelvic floor aperture; and if the visceral activity be sufficiently great, the advancing extremity of the mass is insinuated into the pelvic floor aperture, and propelled onwards by the visceral activity, it dilates the aperture in a wedge-shaped sort of way, and is passed through. Usually, the activity of the viscus concerned, however good it may be, is insufficient for this —even for ordinary defæcation some straining is necessary. This may amount to but a momentary holding of the breath, accompanied by a similarly brief depressing movement—when peristalsis is good, such usually suffices. But when peristalsis is poor or absent, the reverse is the case: straining becomes then the dominant factor. Under such conditions, straining is usually considerable and often great; and is often prolonged and repeated several times. The reason of this is that expulsion depends primarily on the visceral musculature; and when this is inactive and straining takes its place, the greater the

straining the greater the resistance of the pelvic floor, and the greater is the difficulty of forcing the body to be extruded into the pelvic floor aperture. Further, straining presses down the bladder and vagina on to the rectum, and in the absence of rectal activity easily obstructs the fæcal advance. Thus, we see how it is that in such cases the effort expended is so often so much out of proportion to the effect produced and end desired.

Whilst the degree of straining varies inversely with the visceral activity, it also varies with the volume—the calibre—of the body to be passed: the greater this is, the greater the straining. This is so even when the activity of the viscus concerned is good; but it occurs at a later stage, when the body to be passed has already entered the aperture. And although the cause—the resistance to dilatation of the pelvic floor aperture by the pubo-rectalis—is the same in this case, to it must be added in some cases the resistance to dilatation of more superficial parts (e.g. of the vaginal and vulvar outlets during childbirth).

Clearly, when the body to be passed has begun to enter the aperture, straining will be helpful; for it will force the body farther into the aperture and forcibly dilate the aperture by exerting a laterally expanding pressure, until the aperture is large enough to accommodate the largest circumference of the body being extruded. Even though the resistance of the pelvic floor is increased by the straining which causes this descent and dilatation of the aperture, the musculature about the aperture (pubo-rectalis), being now at a disadvantage, cannot prevent this dilatation if the straining be great enough. In the case of defæcation, when the necessary dilatation has occurred, the straining naturally subsides and the resistance of the pelvic floor correspondingly reduces. The aperture

having been sufficiently dilated, the fæcal mass within it, maintains it in this state; and all that is necessary is to force the mass through. Thus, as the act occurs, the straining is reduced to a degree sufficient for this; so that completion is easy.

The same happens during parturition. But in first cases, owing to the deflection of the anterior pole of the head forwards by the stretched perineal tissues, the aperture is dilated more than is necessary—because an ellipse of the head, rather than a circle, occupies the aperture. Since this movement is caused to a large extent by straining; since with straining the resistance (the contraction) of the pelvic floor musculature increases; it is easy to see how in such cases the muscle is more liable to injury than when an early perineal tear occurs, or when extension of the head forwards is prevented by traction of the fingers. Not only is the tonically contracted muscle less stretched; but birth can occur with a lesser degree of straining. Similarly, the use of anæsthesia at this stage of labour tends to spare the muscle from injury: for it reduces the straining, prevents violent and sudden efforts being made, and by delaying expulsion, allows extraction to become a much more governable affair.

Thus, straining is only of service when the body to be passed has already begun to enter the aperture; straining then assists the movement. So also, the larger the mass being extruded, the greater is the effect of straining. But before the mass to be passed has entered the aperture, straining is either altogether abortive or renders the act much more difficult; for it only increases the resistance of the pelvic floor and tends to prevent the entry of the body into the embrace of the pubo-rectalis.

'To assist then the activity of the viscus concerned, a variable increase of pressure often considerable has to

be produced within the abdomen and brought to bear on the pelvic floor, especially anteriorly in the region of the aperture. It is to produce this increase readily and to direct it properly, so that defæcation for instance may be accomplished as easily as possible, that the crouching attitude is assumed. By flexing the spine, the pubes and thorax approach and the cavities of the abdomen and pelvis are brought more into line. But besides this, the anterior abdominal wall is bulged; and moreover, is shortened from above downwards so that the fibres of the flank muscles are crowded together. Thus, the very best conditions for a high pelvic pressure are produced; and any increase that may arise is directed much more anteriorly on to the pelvic floor than could occur in other positions. A breath being now taken and the glottis closed, the diaphragm being thus fixed at its lowest point, the musculatures enclosing the abdomino-pelvic visceral mass all contract, whilst the flexion of the spine may be simultaneously increased. Thus the pressure is greatly raised and the increase is directed into the pelvis, and presses the collective pelvic viscera down on to the pelvic floor with sufficient force to slightly depress it and to correspondingly dilate its aperture. This occurs in spite of the contraction of the pelvic floor, which is not only continued but actually increased —explaining the effort to be made, and the small amount of depression which occurs. The aperture being somewhat dilated by this movement, whatever is immediately over the aperture tends to enter it. If this consists of the conical extremity of a fæcal mass tending to enter the aperture because of the activity of the gut, its entry occurs; and the further dilatation of the aperture is easily completed. If it consists of the caput succedaneum, this also to some extent can enter the aperture and begin to exert a laterally expanding pressure on the

pubo-rectalis. But if it consist of a hard, spherical superficies of considerable diameter, such as the bony head of the child before deformation has occurred and a caput has formed, such entry cannot happen and delivery is delayed until such changes have had time to be produced.

And that the crouching attitude is necessary in man for defæcation—that it is not merely a convenience—is shown by what happens when the individual determines to postpone the act. In this case, the very opposite conditions to those obtaining for facilitating it, are produced. The posture assumed is observed best when defæcation is urgent, and yet must be postponed. Under such circumstances the spine is maximally extended, and the thorax expanded as much as possible so as to reduce to a minimum the intra-abdominal and so the pelvic pressure: the recession and obliquity of the pelvis becomes maximal to minimise the effect of the visceral pressure on the pelvic floor, especially on the pelvic floor aperture. And instead of being direct, the impact of any increased visceral thrust (as produced by movement) is indirect; it only reaches the floor by reflection and diffusion. With this, a voluntary maximal contraction of the pelvic floor musculature is produced, in the hope of effectually constricting the rectum. This is necessary: for when the intra-abdominal pressure is reduced, the tonic contraction of the pelvic floor musculature is correspondingly diminished; and thus, when peristalsis is violent and especially when the rectal contents are fluid, the musculature, unless voluntarily contracted, is easily overcome. Indeed, in such cases, in spite of a contraction as great as possible, the musculature may be unable to prevent fæcal escape. Its function, as we have seen, is not to constrict the rectum *per se*; but to maintain the pressure in the pelvis, and its effect on the visceral canals is secondary to this. Under these conditions it is probable, that

the individual by his voluntary efforts, however great these be, is unable to produce such a contraction of the musculature as can easily be produced at other times by reflex action; and it is not surprising that the visceral musculature often should triumph.

The influence of the crouching attitude in rendering defæcation easy, is seen, too, in the difficulty experienced in performing the act in other positions. Thus, in the dorsal decubitus, defæcation is often difficult—because, in this position, a high pelvic pressure is difficult to produce and to maintain. This opinion—that a high pelvic pressure is necessary and is occasioned by the persistent activity of the pelvic floor musculature during the act, especially of that part of it guarding the pelvic floor aperture—is confirmed by a study of comparative defæcation and anatomy. Thus, in the dog and the horse, both pronograde mammals, the postures assumed are quite different. For the dog, crouching as in man is always necessary (or nearly always, except when the fæces are fluid): but in the horse crouching never occurs! The reason of the difference is that the dog possesses a pelvic floor musculature comparable with that of man, which has to be overcome, that is stretched, the aperture has to be dilated by the fæces before they can pass (see Fig. 7, p. 29): but the horse possesses no such mechanism obstructing the fæcal mass—no such muscle homologous with the levator ani of the dog (pubo-coccygeus) being present in this animal.

2. The Retention of the Viscera during Straining

Such considerations show us plainly enough that during defæcation the pelvic floor musculature still functions—and, indeed, to an exaggerated degree: and, it is clear, the same happens during straining—i.e. even when no extrusion results. But whilst the visceral retention during

defæcation is easily intelligible; during straining, it is not at first sight plain. In the former case, the fæcal mass during its extrusion prevents any tendency for visceral escape. The aperture, as we have seen, is not dilated passively (by muscular inhibition); but actively, by the fæcal mass depressed by the peristalsis and straining. To be extruded, the fæcal mass, if of ordinary size and consistency, not only has to distend the anal canal, but the aperture itself. It has, as a rule, to dilate the pelvic floor aperture to a greater extent than straining alone—i.e. in the absence of such a dilator—can cause. Thus, the fæcal mass, by dilating the aperture, acts for the time being as an obturator, occluding the aperture, rendering impossible any visceral escape.

But when straining occurs apart from attempted defæcation (as during examination), or apart from sufficient visceral activity, the conditions are different. In this case, the fæcal mass is not directed forwards towards the anal canal, and not pressed downwards into the aperture dilated by the straining. The visceral parts immediately over the aperture are themselves pressed downwards into the aperture. Why, then, does their extrusion—their prolapse—not occur?

It is plain, the visceral mass itself, under these conditions, must participate in the occlusion of the aperture. It must do so either by means of its connective tissue attachments —an idea we have already dismissed; or "fixation" occurs in the other way we have considered. Clearly, during the straining associated with visceral activity and causing a fæcal (or ovular) extrusion, an anchoring of the organs to the pelvic walls is out of the question. Indeed, the parts immediately superjacent to the aperture—the median part of the bladder base and the part of the vagina normally related to the aperture—so far from being suspended, are pressed down on the distended perineal flexure and upper

extremity of the anal canal (or upon the dilated vagina) and assist in the action. Thus, the existence of ligamentous strands of connective tissue or of pedicles, however dense or strong, for suspending the viscera, is plainly unnecessary. Similarly, these connections for such an end are unnecessary for the retention of the viscera in the pelvis during the straining occurring apart from visceral activity.

The latter alternative is the true one. The changes in the visceral mass, produced by increase of pressure —with which increase, the activity of the pelvic floor is plainly concerned—explain the retention. A parallel with resting states in the normal can be drawn. For just as the uterus during non-straining times is maintained in position by the resistance to displacement of the surrounding parts; by the resistance of the walls and floor of the pelvis; and by the compression from above; so it is plain that during straining these three factors are still present. And it is obvious that the increased intra-abdominal pressure must have some effect on the position of the uterus during straining, just as it does at other times; that it is instrumental either in maintaining the organ in the new position it has acquired as the result of the straining, or else in changing that position for some other position by further displacement of surrounding parts, by still further depressing the pelvic floor, or by causing a still further enlargement of the aperture—with some possible visceral extrusion.

That such does not occur depends on the integrity of the pelvic floor, and especially of that part of it about the aperture. Between the resisting pelvic floor below and the greatly contracted thoracic diaphragm and abdominal wall above, all the viscera are correspondingly compressed; they are all pressed against each other and against the roof, walls, and floor of the container with an added force. Those in the pelvis are compressed so much against the

pelvic walls and floor that they have caused the floor itself to descend before the pressure, and especially are those lying immediately above the aperture pressed downwards upon it and its margins, causing with their descent a dilatation of the aperture. It is the pressure of these parts, related to the aperture and which overlap the aperture, which is especially important. In a few cases, this pressure (the condition causing it being frequently repeated) causes an extrusion of these parts; in the majority, it fails to do so—according to the size of the aperture the straining determines in the respective cases; according, that is, to the resistance the guardian muscle about the aperture is capable of displaying during this time of stress.

The mechanism of prolapse is easily intelligible. On straining, in the presence of a damaged pubo-coccygeus * and a damaged pubo-rectalis, the visceral parts superjacent to the aperture are bulged into the aperture, and the larger the aperture the greater this initial bulging. The greater the bulging, the greater the descent; and the more these parts can enter the aperture the more can they exert an expanding pressure circumferentially on the boundaries of the aperture, tending to enlarge it according to the pressure transmitted to them from the pelvis.

But when the aperture is not much enlarged by straining, as is the case under normal conditions, extrusion does not occur. Medianly, just above the aperture, the parts are bulged into the aperture, but not to the same extent as before. But the same pressure which causes the bulging, besides pressing all the visceral parts in the pelvis more tightly together, presses those parts of the viscera affected, and which overlap the aperture, on to the resisting parts of the pelvic superficies round and about the aperture, its margins and adjacent parts, with considerable force. Whilst

* The injury of childbirth is usually or for the most part unilateral.

the median basal part of the bladder, bulging the related part of the anterior vaginal wall before it, is pressed into the aperture and against the resisting margins of the aperture; the parts of the bladder circumferentially to this are pressed downwards on to surrounding parts—on to the pubic arch in front; on to the pubo-coccygei at the sides; on to the vagina and rectum posteriorly, which in turn are related to and supported by the pelvic floor posteriorly to the aperture. It is the resistance of these parts which suffices to retain the viscera in the pelvis during straining, in spite of the aperture being somewhat dilated, and in spite of the considerable pressure within the pelvis which tends to force the median parts downward and does in fact bulge them into the aperture. The resistance of the pelvic floor, increased by the straining, is able to do this, because with the straining the deformability of the visceral mass, for the time being, is diminished or even abolished. The visceral mass related to the pelvic floor, becoming (with the other parts of this mass) relatively indeformable, presents, for the time being, a non-yielding platter-like superficies (bulged in the region of the aperture) which effectually obliterates or occludes the aperture.

We have already seen that the intra-abdominal pressure when considerably increased causes fixation of the organs: that it should do so during straining is not extraordinary. The increased pressure with which all the organs are pressed together and against the walls and roof and floor of the compressing container, obviously renders less easy the displaceability of the component parts, than when the general compression is lower—rendering them less easily deformable—even such hollow organs as the intestines. And that such fixation plays a part during straining in the retention of visceral parts must also be allowed.

Were the visceral parts and the connective tissue (the mesodermal matrix) about the organs easily deformable or displaceable during straining; did the medium in which the uterus is poised and compressed consist of easily displaceable particles as those of a mobile liquid; then, indeed, would they all be "prolapsed" during straining. But it is not so. The important part played in this retention by the pelvic floor has been referred to; the condition of the visceral parts themselves is plainly a factor. When the pelvic floor is intact and its aperture is not too much enlarged by straining, the visceral parts, rendered relatively indeformable or much less deformable than before, come into play. That they can do so is conditioned by the brief duration of straining; that they do so is indicated by cases of partial prolapse to which we shall refer—the persistence of the "partial" form; and that the mechanism is possible is evident from physical experiment.

The briefness of the duration of straining is of great importance. During straining the unsupported parts of the anterior vaginal wall and bladder are exposed to a very considerable pressure, which is the greater the effort expended; and however good be their tone (e.g. of their epithelial cells), their tissues naturally tend to yield and to stretch as its result. But this takes time, and the duration of straining is so short that these parts suffer little if at all. If their tone should become impaired, it is regained subsequently, and that is the reason why straining can occur again and again without prolapse arising. It is only in women in whom the pubo-rectalis has suffered from childbirth, yet insufficiently to allow the exciting cause of prolapse such as occurs with ordinary activities to be effective in causing an extrusion, that the frequency of straining matters. In them the frequency of the act compensates for its short duration. The parts of the vagina and bladder unsupported are now more extensive than

under normal conditions; the pressure from above has a greater opportunity of depressing them, for it bears upon a larger unprotected surface: their tissues become more readily affected and their stretching more easily occurs. Subsequently to the straining they are not so supported as in the normal; and if the period of rest is not correspondingly prolonged, they do not regain their tone upon which their normal position depends. Thus, the stretching begun during straining persists, and with repetition of the process becomes greater.

As we have seen, when straining is associated with successful defæcation, this depression of visceral parts immediately above the aperture and their stretching does not occur. During the passage of the fæcal mass, these parts are not unsupported; they are pressed downwards on the distended gut and other parts forming the obturator. It is only when straining occurs apart from this that it is pernicious. Especially in women with a larger aperture than normal should such straining be avoided; for if often repeated it is most likely to cause the symptoms of prolapse, and in time even an extrusion itself.

That viscid liquids in virtue of their viscosity cannot pass easily through apertures smaller than a certain size is known to all. And that the visceral mass abutting upon the aperture will itself present an obstacle to its passage for such a reason is not difficult to understand: much more must this do so if its viscosity is increased (or its deformability is diminished) by increase of pressure—even though such increase of pressure will tend to cause an extrusion. We have indeed evidence that under somewhat similar but experimental conditions, increase of pressure not only is unable to cause an "extrusion" (the aperture being small enough), but actually is the cause of the "retention" which occurs.

Thus a piece of paper, thin enough to be easily crumpled, placed on a board with a circular hole or oval opening, comparable to that of the pelvic floor aperture, can be kept in place by air pressure applied to it provided enough of it overlap the orifice. If the hole be too big, or if the paper is not large enough to overlap the orifice sufficiently so that the pressure can fix it to the circumferential supporting surface, the paper gives way in some direction and bulges through the hole. Professor Tandler demonstrated this some years ago to his class when I happened to be there. But in this case, no sphincteric mechanism is in play. In the pelvis such is present, exerting a restraining influence even during straining; whilst the tensile strength of the paper used in the experiment, is replaced by the increased viscosity of the tissues forming the plastic pelvic pole of the visceral mass.

CHAPTER XI

THE PELVIC FLOOR AND THE VISCERAL MASS IN CASES OF PROLAPSE

1. The Pathological Changes in Partial Prolapse and their Significance

The conditions found in prolapse, especially the persistence of the "partial" form, and the changes which arise with the advance of the disease, support this conception. Clearly, unless the visceral mass, in virtue of its physical peculiarities, blocks the aperture and so retains the viscera remaining in the pelvis, cases of "partial" prolapse could not occur. In the absence of such a provision, not only would straining in the normal be disastrous; but in cases in which prolapse is imminent, the moment extrusion began it would rapidly become "complete." For just as the visceral connective tissue hypothesis, as an explanation of the retention of the viscera during straining, in spite of its plausibility, is invalid; so, applied to cases of prolapse, it is equally untenable. It does not explain the persistence of the "partial" form; nor the advance of the disease. This is stayed by other forces than are implied by that hypothesis—by just those, indeed, by which extrusion during straining in the normal is prevented: (1) by the activity of the pelvic floor; (2) by the physical features of the visceral mass; and (3) by the prevention of too high pressures in the pelvis, which if too long sustained or too often repeated, could not be opposed.

It is not surprising, in the presence of an increased pressure in the pelvis due to activity, that the visceral mass

related to the pelvic floor aperture, enlarged by injury, should bulge into the aperture; and that in time a protrusion should come to persist as long as the pressure in the pelvis is raised. Nor is it surprising, since this protrusion is the result of pressure, that changes in both the parts concerned should occur in adaptation to the new conditions.

The pelvic floor aperture is early affected. The parts prolapsed, in virtue of their volume and the pressures within them, stretch the margins of the aperture—the remains of the pubo-rectalis and the adjacent parts of the pelvic floor. By them, the aperture becomes secondarily dilated, allowing more and more visceral parts to "come down"; and this continues until an equilibrium is established which depends especially on the condition of the pubo-rectalis and the manner in which it is able to react to these changes.* In late stages of the disease, this muscle, with the rest of the pelvic floor, is markedly atrophic (Fig. 3, p. 18)—the result of the now large volume of the prolapse and of the greater and extending effect this has had with its increase on the pelvic floor. But in earlier stages, when the prolapse is smaller, what remains of the musculature is often well developed (Fig. 25, p. 351). Indeed, an adaptive response may have occurred so that the aperture has become again smaller. To such an extent is this so in many cases of prolapse, that the condition of the musculature has been brought forward as a reason for believing that it plays no part in the normal retention of the viscera within the pelvis; for if so, in prolapse, it is argued, it ought to be poorly developed. Moritz, for instance, advances such an argument.†

* See Chap. XIV.

† Moritz writes as follows: " . . . I had the good fortune recently to perform a complete *post-mortem* examination on a female with procidentia. Fully two-thirds of the uterus, which was large, lay outside the body and the remaining third was in the vagina. The parametrium was stretched into two long ribbon-like folds; it was

But in these cases, the pubo-rectalis although well developed has lost its former attachments, whereby a narrow aperture obtains, and the ones it now possesses give to the aperture a much larger transverse diameter—the attachments, from being anterior, have become more lateral; or if attached anteriorly, it is by fibrous tissue. Thus, if the prolapsed part is reduced into the pelvis, it recurs again on straining; for although on reduction of the prolapse a diminution of the aperture occurs, the reduction does not allow a return to the normal condition.

The resistance of the pelvic floor to this secondary (and early) dilatation of its aperture, and more especially when the musculature has been able to react and adapt itself to the prolapse—when, therefore, it is in good condition—necessarily reacts on the prolapse itself. When the prolapse is "down," its neck is embraced by the margins of the aperture; and according to the pressure in the pelvis (i.e. to the activity of the individual) and the degree of contraction of the pelvic floor which varies with that pressure, is more or is less constricted by them. The recognition of this constriction is important. That the pelvic floor should play this part under pathological conditions, that is to say, when it has been injured by childbirth and an extrusion has occurred, confirms and emphasises the rôle imputed to it in the normal—even during straining. That it does so is shown by the pathological changes produced in prolapse. To begin with, a passive congestion in the parts extruded is produced; for the

thin and scanty. The pelvic diaphragm was healthy and normal. Beyond an increase in the size of the vulvar orifice, obviously mechanical in origin, no pathological changes existed. The musculature of the pelvic diaphragm *was well developed.* I have satisfied myself, and have specimens to demonstrate, that efficient colporrhaphies are successful only because they tighten up the parametrium, though some surgeons who practise these operations may not quite realise this" [66].

constriction interferes with the passage of blood, impeding especially the venous return. In this way, the nutrition of the prolapsed parts is impaired: œdema of the prolapse and fibrosis occur; and often ulceration.

But more conspicuous internal changes are found. Thus, when the bladder is partially prolapsed and contents are present, the organ is distorted into an hour-glass shape (Fig. 20). The part still within the pelvis is bulged by the urine, and the cystocele is similarly enlarged; but the part within the pelvic floor aperture is constricted, being the smallest part of all (Halban and Tandler). The uterus, when partially prolapsed, is similarly affected. Whilst the body of the uterus may be of normal size, the cervix is enlarged from passive congestion and its results; but the part between, within the embrace of the aperture, is thinned by constriction. Halban and Tandler emphasise this relationship: the constricted part of the bladder in cystocele, and of the uterus when partially prolapsed, is always at the level of the "genital hiatus"—the name these authors give to the "cleft" between the levator muscles, called in this book the "pelvic floor aperture."

Explanatory Notes (Fig. 20)

The Coccyx and the post-anal part of the Pelvic Floor occupy their normal positions. The post-anal part of the floor is much shortened by the increase of the aperture antero-posteriorly. Thus, the distance from the lower border of the symphysis to the perineal flexure of the gut is much greater than normal. (According to Halban and Tandler, the pelvic floor in this case was on the whole well developed, the white line a little broader than normal and displaced a little downwards. The drawing they give of the floor shows a condition intermediate between the normal and that depicted in Fig. 3, p. 18.)

The mass prolapsed is constricted at its neck, in the region of the pelvic floor aperture; so that the bladder and uterus together form an hour-glass shape. The constriction of the bladder is well shown; that of the uterus is not so marked.

Note the position of the ureteric orifice: it is placed well below the region of the constriction, and it is intelligible how the ureter itself in the region of the neck of the prolapse must suffer compression. The urethra is distorted and dilated; and it is plain that distension of the cystocele by urine could easily compress the urethra against the pubic arch and render micturition difficult.

The anterior vaginal wall is completely prolapsed, but the eversion of the posterior vaginal wall is incomplete—the usual condition.

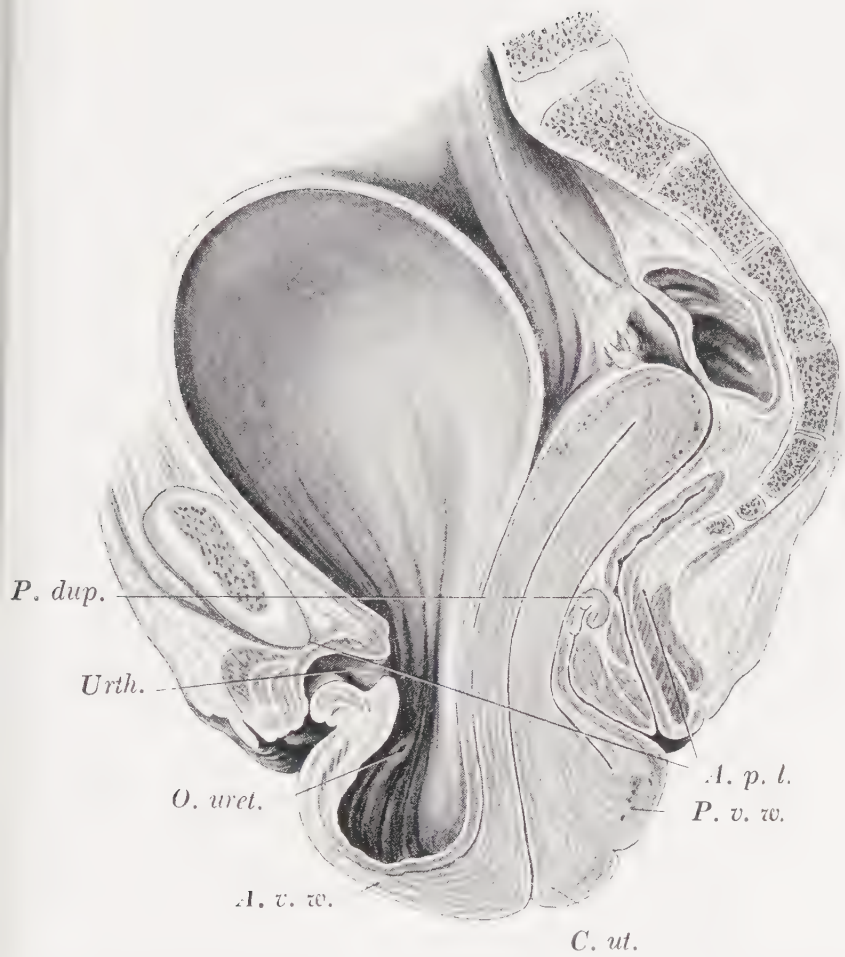

FIG. 20.—A VIEW OF THE RIGHT HALF OF A PELVIS FROM A CASE OF PARTIAL PROLAPSE, SHOWING HOW THE PARTS PROLAPSED PARTICIPATE IN THE OCCLUSION OF THE ENLARGED PELVIC FLOOR APERTURE.

(From Halban and Tandler.)

O. uret. = Ureteric orifice.
A. v. w. = Anterior vaginal wall.
C. ut. = Cervix uteri.
P. v. w. = Posterior vaginal wall.
A. p. l. = Antero-posterior limits of the pelvic floor aperture.
P. dup. = Position of the duplication of the posterior vaginal wall.
Urth. = Urethra.

(*For Notes see opposite page.*)

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According to them, the thinning of the uterus in this region is especially marked in the antero-posterior diameter, the uterus here measuring in some cases only half a centimetre thick. In the literature, they found that even a separation of the body of the uterus from the cervix, the result of such thinning, has been described (v. Franque, [30]).

But perhaps the effect on the ureters shows best this activity of the pelvic floor in cases of prolapse (Fig. 21). Since with the occurrence of cystocele, the distal parts of the ureters are extruded,* the ureters as the condition advances come to pass through the pelvic floor aperture, and like the bladder itself to be compressed there. Halban and Tandler tell us that in many cases of cystocele the proximal parts of the ureters are found dilated and hypertrophied ; and that this so far from being an exceptional change, is typical. Out of 23 cases examined, these authors found a pronounced dilatation of the ureters fifteen times. In some cases, the ureters were of the size of a finger. And the limit between the dilated and the non-dilated parts corresponded exactly with the margin of the " genital hiatus " (Halban and Tandler). According to these authors, the dilatation of the ureters is produced in an entirely mechanical way—by the damming effect of the " hernial ring " where the channels are constricted.† Only in those cases they examined in which the cystocele was so small that the ureteric orifices were above the " genital hiatus "—that is, were still intra-pelvic—was no hydroureter present. In other cases, they attribute its absence to the patient having worn a pessary during life. But, manifestly, in more advanced cases, there is another reason for this absence. In advanced cases of prolapse,

* Cystocele usually begins with the depression and extrusion of the retro-ureteric recess of the bladder (Halban and Tandler).

† Uræmia has been ascribed to this cause [35].

in which the pelvic floor has become markedly atrophic, constriction of the prolapsed mass by it can no longer occur ; and such dilatation of the ureters if present at an earlier stage of the disease can obviously pass away. Thus we see in Fig. 17 (p. 120,) the ureter is represented as normal in size.

That these changes in partially extruded viscera must be attributed to the effect of the pelvic floor compressing the prolapsed mass in the region of the aperture, must be patent to all. By it, the prolapsed mass is constricted at its neck ; and thus the pressure in the pelvis tends to be maintained, and—if the obturator do not yield—the pressure is maintained. Consequently, visceral parts above, still within the pelvis, and abutting upon the parts prolapsed, stay in position—they can only be prolapsed by depressing the parts forming the obturator (so that other parts come to form the obturator), or by enlarging the obturator. For this to occur the resistance of the pelvic floor compressing the obturator must be overcome. But in cases of partial prolapse in which an equilibrium is established (in which the disease neither advances nor recedes), this resistance is not readily overcome, for in these cases what remains of the pelvic floor is usually in a good condition ; and increases of intra-pelvic pressure, which tend to increase the extrusion are prevented from doing so by its action. The pelvic floor, by preventing the passage through the aperture of non-prolapsed parts, prevents the obturator from becoming enlarged or depressed.

2. The Fixation of the Neck of the Prolapse in Cases of Partial Prolapse

That such a mechanism is in play in cases of partial prolapse, is plainly evident. In solitary extrusion of the

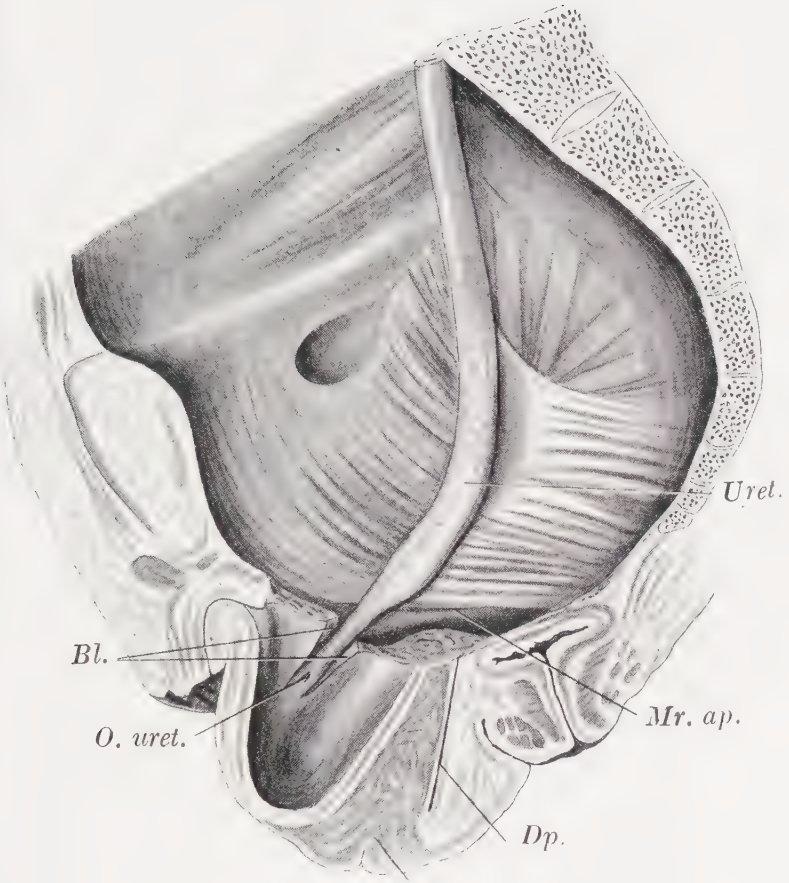

FIG. 21.—A VIEW OF THE RIGHT HALF OF A PELVIS DISSECTED TO SHOW THE DILATATION OF THE URETER FROM A CASE OF PARTIAL PROLAPSE IN WHICH THE BLADDER AND UTERUS WERE INVOLVED.

(From Halban and Tandler.)

The ureter is displayed throughout its lower pelvic part, and its relation to the pelvic floor aperture is seen. Whilst the part prolapsed is of normal size, that above the level of the margin of the aperture is dilated.

Hart and Barbour refer to this compression of the ureters in prolapse, attributing it to pressure of the pubic arch. [35]

Bl. = The retracted margins of bladder wall after incision to display the terminal part of the ureter.
O. uret. = Ureteric orifice.
C. ut. = Cervix uteri.
D. p. = Douglas' pouch.
Mr. ap. = Margin of the pelvic floor aperture.
Uret. = Ureter.

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cervix, the reason more of the uterus is not prolapsed is that the organ, with the walls of the visceral canals occupying the aperture, efficiently blocks the aperture—when the pressure is low, it does so easily; when the pressure is raised, the occlusion is correspondingly firm. The pathologically enlarged obturator is sufficiently embraced by the margins of the aperture to prevent further extrusion. On the one hand, the uterus, the fundus of which projects into the pelvis, tends to be depressed by the pressure about it: on the other, the pressure relations between the part of it embraced in the aperture and the margins of the aperture is great enough to prevent the former force from becoming effective. The maintenance of the *status quo* depends, then, on the activity of the pelvic floor musculature, and on the resistance of the uterus: nor does the fact that the uterus is separated from the margins of the aperture by the vaginal walls and rectum—i.e. by surfaces between which and the uterus gliding movements could easily occur when the pressure is low—prevent such maintenance. That pressure contact can keep such opposing surfaces in position, in spite of the presence of forces tending to displace the one on the other, will be admitted.

This is the simplest case to consider. For the uterus is a more or less solid organ; and it is easily intelligible how it can act in this way—doing so in virtue of its indeformability, which is conspicuous even when the pressure about it is low. But in other cases of partial prolapse the condition is complicated by the hollowness of the organs involved in the prolapse; and, in the case of the bladder, by the mobility of its contents. In cases of cystocele, it is plain, if the part of the bladder embraced by the margins of the aperture is constricted sufficiently to cut off the intra-pelvic part from the part prolapsed (so that urine cannot pass from the one to the other) that the part of the bladder embraced will be maintained in position. If held

in this way, no increase in the extrusion will occur. But if, on the other hand, the constriction is insufficient for this, pressure variations by way of the urine will easily be transmitted to the parts prolapsed and can only be resisted by the tensile strength of the "coverings."

At first sight, one is rather inclined to attribute to this a considerable rôle in the prevention of further extrusion. Clearly, if, in the case depicted in Fig. 20, the anterior vaginal wall and the related part of the bladder gave way, the urine distending the bladder would escape, and unless the pelvic floor aperture became correspondingly smaller, more of the uterus and upper part of the bladder would be extruded. On the other hand, Halban and Tandler have shown, in the recently dead, in which cystocele and partial prolapse with elongation of the uterus were present, that by distending the bladder the whole prolapsed mass spontaneously reduced itself into the pelvis [34]. It can be easily understood how (in the dead) with great distension of the bladder, the part prolapsed should be withdrawn into the pelvis, and how the entire organ should act as a ball-valve blocking the enlarged pelvic floor aperture—for with such distension, the bladder becomes markedly tense and much less deformable than before. But in the living, if equilibrium is to be maintained, the parts prolapsed play a different rôle. They do not act in virtue of a tension of their walls; but in virtue of their mass and incompressibility. Fig. 20 probably represents a transition from the partial to the complete form: the condition shown in Fig. 25 is probably the actual state of affairs when the partial form persists.

It is impossible to think that in cystocele, the parts prolapsed, in virtue of an increase in thickness, from which an increased resistance to tension results, themselves prevent the prolapse advancing: that they are capable of sustaining

the pressure and its changes which occur in the pelvis with healthy activity. They could only do this if the neck of the prolapse were sufficiently suspended ; and if the parts below were sufficiently resistant. But in many cases of cystocele the part of the bladder prolapsed is extremely thin ; and the thickening which the vaginal wall gradually undergoes is conspicuous only in late stages of the disease. It is only in advanced cases of prolapse, in which the prolapse is " complete " and the pelvic floor has become so atrophied that it can no longer constrict the base of the prolapse, that the superficial parts of the prolapse come into play, sustaining and resisting such pressures as occur. In such cases the extruded mass is remarkable for the density of its peripheral constituents. This change is comparable with the thickening occurring in the sac of any other hernia which has remained " down " for any length of time (e.g. in scrotal hernia) ; and it is due in the main to a similar cause—the increased strain to which the connective and other tissue cells are subjected owing to the prolapsed mass being no longer supported by the parietes and having of itself to bear the strain of the intra-abdominal pressure and of its excursions. But the pressures occurring in the pelvis in these cases are not comparable with those produced in the normally active woman—high pressure changes cannot occur, for the simple reason that the mechanism occluding the pelvic outlet is now out of play.

Nor would the changes which the prolapsed parts in partial cases undergo occur, at least to the extent they do, unless a very considerable constriction of the parts prolapsed in the region of the aperture was more or less persistently maintained. Especially are the changes in the ureters significant in this respect. If the constriction were insufficient to prevent the transmission of pressure from the pelvis to the prolapse—if the parts prolapsed (in cases of partial prolapse) sustained the pelvic pressures—the

ureters would remain unaffected. For the urine, which is secreted into the part of the bladder prolapsed, would easily flow into the intra-pelvic part; and in time be naturally voided. The ureters would have but to propel the urine onward with a force sufficient to overcome the pressure in the bladder—a pressure no greater than in the normal. The fact that the ureters become dilated shows that there is an obstruction to the urine in the ureters. That the limit between the dilated and undilated parts occurs at the level of the pelvic floor aperture shows that it is here that the obstruction exists.

Similarly, the difficulty of micturition complained of by many of these patients is probably due, at least in some cases, to this constriction. It frequently happens that the individual herself has to replace the cystocele before micturition can occur; or, to assume a peculiar posture—for instance to stand. In the former case (which shows how little the prolapsed wall of the bladder contracts), the proximal part of the urethra with the cystocele is replaced in the pelvis, and a free communication with the intra-pelvic part of the bladder established—before this is effected, either the pressure in the intra-pelvic part of the bladder cannot be communicated to the cystocele, or it cannot dilate the urethra. Since on straining the pelvic floor aperture is dilated, the former possibility is unlikely to obtain, and it is more probable that the pressure in the cystocele (due to the accumulation of urine and to the straining) causes the cystocele to compress the urethra against the pubic arch (see Fig. 20). The change in position facilitating micturition is more significant; for in this case a change in the pressure conditions is produced whereby the contraction of the pelvic floor is reduced and the act rendered more easy. On standing the pressure in the pelvis is less than when crouching; and thus the contraction of the pelvic floor—and so the constriction of the

neck of the prolapse—is less in the former than in the latter position. It is obvious that in these cases straining scarcely ever or never occurs.

The visceral tissues stretch so easily, and when unsupported are so unsuited to sustaining pressures, that such a mechanism—their resistance to tension—in the prevention of further extrusion cannot obtain. If a partial prolapse is to persist, the parts prolapsed must be preserved to a very great extent from the pressures produced in the pelvis. The only way in which this can happen is by the neck of the prolapse being sufficiently embraced to prevent the transmission of pressure changes from the pelvis to the prolapsed parts. That is to say, that the mechanism by which the prolapsed parts in cystocele are maintained in position is identical with that by which the uterus, in cases of its partial (and solitary) prolapse, is sustained.

We may then reasonably suppose, in cases of persistent partial prolapse, that the constriction of the neck of the prolapse is great enough to prevent the transference of pressure changes to the prolapse —especially during static or active states when the pressure in the pelvis is fairly high. If the pelvic floor musculature is sufficiently adapted, if it can contract sufficiently powerfully and for as long as is necessary, equilibrium will be maintained. If, however, the pressure in the pelvis is too great or is continued too long, the muscle in time yields ; the neck of the prolapse becomes less constricted ; the pressure between it and the margins of the aperture falls ; and parts within the pelvis abutting upon the aperture tend to be pressed through, bearing down pains and a sense of weakness being experienced.

But even when the best possible response of the muscle has occurred, and it is able to successfully oppose pressures due to static and active states, the constriction it causes is

not great enough to continuously maintain this state of affairs. With great and sudden rises of pressure, the pelvic floor yields in front of each impact with a temporary enlargement of the aperture and stretching of the prolapsed parts (e.g. on cough). The pelvic floor, though adapted to the prolapse, is not good enough to oppose such rises, any more than in the normal. But with the impact and expansion of the obturator and stretching of the muscular margins of the aperture, an increased contraction of these muscular parts occurs. Thus, the obturator is grasped more firmly; and thus, its position tends to be maintained. The pressure between the obturator and the margins of the pelvic floor aperture, instead of falling becomes increased, so that fixation persists. It is only when such impacts occur too frequently, and the muscle is unable to respond (by a reflex contraction) towards them in a series of too rapid sequence, that an increase in the prolapse tends to occur. During such changes—the musculature being efficient—it is plain that the parts prolapsed participate in preventing further visceral extrusion, just as they do in the normal during straining; but, just as in that case, they can only do so temporarily. And this—in cases of partial prolapse—is the only condition under which the tensile strength of the parts prolapsed comes into prominence, preventing further extrusion.

3. The Intra-Pelvic Organs and the Connective Tissue Attachments in Prolapse

Thus, it is an error to suppose that in partial prolapse the parts prolapsed are suspended by the intra-pelvic parts of affected viscera; and that the prolapse advances because the prolapsed parts drag down more of the viscera. We cannot believe that in solitary extrusion of the cervix, the elongation of the uterus is caused by the traction downwards

of the cervix (that the elongation is an effect of gravity); nor that in cystocele, the intra-pelvic part of the bladder suspends the part prolapsed. The mass of the cervix—small before its secondary increase, due to œdema, etc.—puts the former suggestion out of count; the difference in the condition of the part of the bladder prolapsed from that of the part still within the pelvis renders the other untenable.

Examination of these two segments of the bladder in cystocele shows that whilst the wall of the part prolapsed is so stretched that its lining is smooth, the intra-pelvic part is unaffected in this respect. That this should be so is easily explained. For even though the pressure within the intra-pelvic part of the bladder is no higher than that within the part prolapsed, since the former part is pressed by adjacent viscera against the superficies of the pelvis (including remains of its floor), the pressure within it is ineffective in causing a stretching of its walls—a high pressure can exist within, without smoothing out the creases in its mucous lining. But in the part prolapsed, the conditions are different. This part is pressed against the everted anterior vaginal wall, and perhaps the adjacent part of the protruded cervix: but these, being unfitted for the sustaining of pressures, and being themselves unsupported, naturally give way, allowing the affected bladder wall itself to do so.

Thus the idea that the visceral parts within the pelvis suspend those prolapsed is out of the question. Not only are the intra-pelvic parts preserved from being stretched and pulled on by the parts prolapsed (because the parts between, in the level of the aperture, are held in position by the pelvic floor); but when the pressure in the pelvis is raised, these intra-pelvic parts are pressed downwards with greater force upon the remains of the pelvic floor and upon the tissues forming the obturator. But this

obturator simultaneously becomes more tightly compressed; and, if the pelvic floor continue to hold it in position, the prolapse is not increased, in spite of the pressure rise.

It is true that, as the cystocele increases, the parts within the pelvis gradually become shifted. But this is not because the circumferential parts of the bladder are dragged on by the prolapsed mass, but because the pelvic floor aperture becomes larger and larger and more and more of the bladder as of other visceral parts are affected by the expelling force. This, clearly, is the cause of the advance of the displacement. The reason the pelvic floor aperture becomes larger and larger is that the obturator gradually gets bigger and bigger: the reason the obturator increases in size is that the pelvic floor musculature forming the margins of the aperture is gradually more and more overcome. The reason that the pelvic floor is able to resist this tendency to enlargement in some cases, whilst in others it cannot do so, we shall briefly allude to (p. 354).

Similarly, it is an error to suppose that the prolapsed parts are suspended by ligaments; and that the prolapse increases because the ligaments give way. In cases of partial prolapse of the uterus it is plain the connective tissue attachments of the organ have nothing to do with its partial retention. The cervical connective tissue thickenings and attachments of the cervix obviously play no part in keeping the fundus of the uterus within the pelvis; for they are attached to the cervix which is prolapsed, and with the displacement of the cervix have become stretched. Nor do the connections of the fundus suffice to explain its retention. These have been denied by all gynæcologists as of any service in the uterine support; and it is obvious that if such a firm tissue as that of the uterus will yield to

constriction, the round and broad ligaments will stretch from tension. Similarly, just as the uterine pedicles have given way and so become longer, so we believe do the ovarian pedicles behave. Thus, there is no structure whatsoever to which the suspension of the uterine body can be imputed. The pressure within, which presses all the visceral parts, the uterine body amidst them, against each other and against the walls and the still resisting part of the pelvic floor, and the constriction of the prolapsed mass by that part of the floor now forming the margins of the aperture are the only forces remaining, which we are reduced to accept as the cause of the retention.

And the peculiarities of the bladder referred to in cystocele demonstrate that the connective tissue attachments of the bladder have nothing to do with the retention of that part of the bladder which is not prolapsed. There is no relationship between the extent of the part retained and any of the so-called connective tissue attachments of the bladder. On the contrary, the extent of the part retained depends upon the pressure in the pelvis, on the enlargement of the pelvic floor aperture and on whether other visceral parts are blocking the aperture — the differentiation between the part of the bladder retained and that prolapsed occurring always in this region.

And if the parts prolapsed were suspended by their "ligaments," these should be better developed in cases of partial prolapse than when prolapse is complete. As a matter of fact, the reverse is the case. In cases of partial prolapse, the "ligaments," especially the utero-sacral—the ones to which most importance has been attached—are either unaltered, or are elongated and atrophied—so thinned that in most or in many cases they are unrecognisable. Only when prolapse is complete do they become "hypertrophied." And the reason is plain. For when

prolapse is complete, the pelvic floor is quite out of play. It is highly atrophic and can neither restrain the prolapse itself by constricting its neck, nor support the remaining pelvic viscera in position. In the erect posture, all hang suspended by whatever attachments they possess, the thickening of which is intelligible. Just as in enteroptosis (in which the abdominal wall is inefficient), gravity plays an important part in causing the positions of the abdominal viscera ; so in these cases of advanced prolapse gravity is effective in determining the position of pelvic visceral parts.

But in persistent partial cases of prolapse, the pelvic floor is still well developed ; and as long as an equilibrim is maintained, it remains so. Gravity in these cases plays no part in the prolapse. It is the increase of pressure in the pelvis due to activity which causes the extrusion when the woman rises from her bed—not the effect of gravity on the prolapsable parts. A small myoma does not *per se* induce prolapse. The advanced case of prolapse depicted in Fig. 3 was not due to the presence of such a tumour—to the increased weight of the uterus: that is only an accidental feature of the case. Just as in the normal, so in these cases of prolapse the activity of the pelvic floor maintains the viscera—even those prolapsed—in position, and takes the strain off the ligaments which they would otherwise have to bear.

Thus, of the two forces concerned in the retention of viscera in the pelvis in cases of partial prolapse, that of the pelvic floor is far and away the more important. Prolapse only occurs because of a defect in this—the pelvic floor is either injured or the pressure in the pelvis becomes too great for it. And prolapse only advances because the margins of the aperture yield to the pressure with which the bulging visceral mass is thrust into it. At the be-

ginning of the disease, the prolapse can only be stayed by the base of the prolapse increasing in size (as by extrusion of the cervix), until it becomes big enough to stretch the margins of the aperture. It is only when this has happened that the muscular margins of the aperture can become effective in grasping the obturator. The stretching reflexly increases their tone; and only when this happens can an adaptive response on the part of the muscle to the prolapse begin—so that the aperture may become smaller. If the muscle responds and responds adequately, a partial prolapse results; if it fails, the prolapse advances until it becomes "complete."

But the visceral mass itself is also concerned in this mechanism. The part of it still within the pelvis, the part forming the obturator and that the prolapsed mass are all continuous, and with the increase of pressure in the pelvis—the result of the increased contraction of the thoracic diaphragm, abdominal wall and remains of the pelvic floor—the whole becomes less deformable, (with sufficient increase of pressure) even rigid, forming collectively a plug effectually occluding the aperture (Fig. 20). Clearly, the increased compression of the obturator will tend to cause the obturator to become more or less rigid; for it is formed of incompressible tissues, and its deformability depends on the displaceability of its component parts which the increase of pressure above and below prevents. The rise of pressure in the pelvis naturally affects the pressure in the prolapsed mass—this cannot get larger* because of the resistance of its coverings; but even apart from the transmission of pressure from the pelvis, the increased compression of the obturator will cause the pressure in the prolapsed mass to rise. It is clear the parts extruded themselves participate in resisting

* Beyond a certain extent, and for the moment. The increase which occurs, requires time.

the pressures arising in the pelvis. If not sufficiently preserved from these pressures (by the neck of the prolapse being sufficiently constricted), they gradually stretch and thicken; and more and more of the visceral parts come down. These changes only happen slowly, because if the pelvic floor is inadequate to meet the intra-pelvic pressures (which it can only do in the way indicated), the pressure in the pelvis is reflexly reduced. The patient immediately ceases a movement tending to increase the extrusion; or assumes a position relieving the sensation caused. Such as the pressure is, it is obviously because of the physical features of the parts prolapsed—their reaction to increase of pressure whereby they become much less deformable than before—that the *status quo* remains.

CHAPTER XII

THE PELVIC VISCERAL CONNECTIVE TISSUE AND ITS THICKENINGS

1. The Parietal Pelvic Fascia

It is one of the triumphs of modern anatomy that the conception of the connective tissue in the pelvis has become simplified. Formerly, besides a layer lining the pelvis, a complicated visceral layer was also described. Whilst the parietal pelvic fascia was recognised as clothing the muscles, without however being very accurately defined, the visceral pelvic fascia was supposed to consist of membranous or fascial layers, having connections with or arising from the parietal pelvic fascia, passing inwards towards the viscera, dividing into septa on reaching them, and forming sheaths for the organs, supporting or maintaining them in their several positions. With these, the description of the connective tissue in the pelvis ended. Thus, little or no notice was taken of a tissue—the fat-laden connective tissue—which occupies so large a portion of the pelvic cavity [19]; and which, as we have seen, in virtue of its volume and incompressibility participates with the other incompressible tissues in the pelvis in the maintenance of the uterus in its normal position.

But as a result of a series of investigations the old conception of the pelvic fascia has become modified. The idea that the visceral pelvic fascia consists of layers has been abandoned, because such layers do not exist. The layers of connective tissue found in the pelvis—layers, that is,

which exist as sheets and as a rule can be easily demonstrated—are part and parcel of the parietal pelvic fascia. They are strictly parietal [78]. The pelvic fascia "begins and ends with the walls and floor of the cavity and has no direct connection with the viscera. . . . It is always related to muscles, and where these end it is attached to the periosteum of the neighbouring bone" [19]. Thus, the parietal pelvic fascia consists of the layer of fascia covering the internal aspect of the internal obturator muscle, the upper surface of the levator ani and coccygeus, and the internal aspect of the pyriformis. But whilst the layer covering the obturator muscle is continuous inferiorly with that covering the levator ani and coccygeus, posteriorly it ends abruptly at the anterior margin of the great sacro-sciatic notch [19], and thus is not directly continuous with the layer covering the pyriformis. This latter layer passes with its related muscle through the great sacro-sciatic foramen without gaining attachment to the anterior bony border of the foramen.

The parietal pelvic fascia, then, is not one single layer of fascia lining the pelvic cavity. At the origins of the muscles invested—above the level of the obturator internus, behind it in the region of the ischial spine, and in front in the region of the pubic arch, and posteriorly at the margin of the sacrum—the fascial sheets become attached to the bone, and the lining of the pelvis is continued by periosteum. But in spite of this the superficies is incomplete —e.g. in the region of the pelvic floor aperture already considered. It is also incomplete in the region of the obturator canal and in that of the great sacro-sciatic foramen. But just as the pelvic floor aperture is occluded by the visceral canals; so the obturator canal is occluded by a visceral offshoot leaving the pelvis to gain the thigh. So also the great sacro-sciatic foramen is occluded by the pyriformis muscle which passes through the foramen and

by the visceral process accompanying the muscle, which conveys vessels and nerves into and from the gluteal region (see Fig. 14, p. 73).

That such visceral processes, in virtue of their volume and incompressibility, do occlude the apertures through which they pass is shown by their anatomy and the occasional occurrence of hernia through their apertures of exit. We know quite well that the femoral canal, besides containing the femoral artery and vein and lymphatics (and possibly a lymphatic gland), is filled up in the well-developed individual with fatty connective tissue, and that only when a disproportion exists between the volume of the contents of the canal and the capacity of the canal can a femoral hernia develop. If the connective tissue in the femoral canal shrinks, then more subperitoneal tissue is squeezed into it and no harm happens. But if the subperitoneal tissue abutting on the femoral ring is sparse, containing little or no fat, some intestine may be pressed into the ring and get caught. Similarly, a radical cure can only be secured either by filling up the space (as by fattening the patient) or by occluding the aperture by a staple or by other means.

It is the parietal pelvic fascia, and where this stops short the periosteum, which forms the real limit to the pelvic cavity—just as in the abdomen the transversalis fascia (for the most part) forms the inner boundary of its wall. Therefore, when the transversalis fascia is incised, as during laparotomy, the cavity of the abdomen—here occupied by subperitoneal tissue—is opened. But just as in the case of the abdomen, surgeons (almost) universally declare the abdominal cavity is not opened until the peritoneal sac is cut into; so also in the case of the pelvis an analogous position is assumed by those gynæcologists, who instead of recognising the pelvic subperitoneal tissue as part and parcel of the pelvic contents, regard it with parts

of the organs embedded in it as a supporting floor. Such ideas are altogether confusing and misleading. The pelvic cavity is limited by the fascia lining its muscles and the periosteum in between; and all the tissues on the inner side of these clearly comprise contents of the pelvis. Thus, the connective tissue in the pelvis, in which the viscera with invaginations of the peritoneal sac, with their ducts and channels that lead from them, with their blood vessels, lymphatics and nerves, are all embedded, is part of the contents of the pelvis—that is, of the pelvic pole of the visceral mass; and that is how it is that, in virtue of its volume and the pressure to which it is subjected, this connective tissue is capable of affecting the position of the uterus, as are the viscera about the uterus themselves in virtue of the space they occupy in the pelvis and the pressure to which they also are exposed.

2. The Visceral Connective Tissue and the Parietal (or Visceral) Condensations

This tissue we have called the visceral connective tissue. In the adult, it is not a homogeneous tissue as it is in the new-born, but it contains thickenings or condensations. Thus, peripherally, where it abuts on the parietal pelvic fascia, it contains such thickenings. It is also thickened about the blood vessels, the nerves, and the ducts (ureters) in the pelvis; and about the viscera themselves, forming ensheathing layers for the organs, and especially is this so about the cervix. But elsewhere it is a "filmy-looking structure" . . . (on account of the lymph spaces it contains) . . . "which extends from the walls of the pelvis to the viscera and peritoneum. . . . It is sometimes loaded with fat; in other cases, this is absent, and it is then more easily broken down; but in all cases, if the fingers are used for this purpose, it will be found that, while the

subserous tissue gives way easily in most directions, it invariably remains firmly attached to the vessels, and particularly those which are passing from the neighbourhood of the sacrum to the rectum, bladder and . . . uterus. The tissue in this position forms a dense partition on either side, dividing the anterior portion of the pelvis with its viscera from that part which is occupied by the rectum" (Derry) [19].

The peripheral condensations of this tissue of sufficient importance to notice exist in that part of it abutting upon the pelvic floor (especially upon the pubo-coccygei), and that pressed against the lateral pelvic walls in the region of the " white line " and the great sacro-sciatic notch. In the latter region, the condensed tissue, in which the sacral plexus of nerves and pelvic blood vessels are embedded, is adherent to the bone in front (anterior bony margin of the great sacro-sciatic foramen) and postero-laterally is closely applied to the pyriformis sheath. Formerly, it was regarded as parietal pelvic fascia, but really it consists of the parietal portion of the subperitoneal tissue [19]. Externally, it is continuous with the visceral offshoot which quits the pelvis in close relation with the pyriformis ; whilst, internally, it is continuous with the perivascular thickening which passes inwards, forwards and downwards about the uterine vessels.

The condensations related to the pelvic floor and to the pelvic walls more anteriorly are also of interest. It was these which formerly gave rise to the idea of the visceral pelvic fascia. The condensation adherent to the lateral pelvic wall was supposed on being traced inwards to split into layers so as to invest the viscera, and thus to become continuous inferiorly with the condensation related to the pelvic floor with which the basal median parts of the pelvic viscera are related. But the conception of such splitting into layers is untenable. For although the

visceral connective tissue about the viscera is condensed in places, forming definite ensheathing layers, notably about the cervix, . . . " any attempt to give definiteness to such layers is not only artificial, but makes the description unnecessarily complicated and confusing, for the simple reason that these layers, though well marked in the regions named, and also about the rectum, pass gradually into the general mass of subperitoneal tissue which fills (up) the . . . pelvic cavity, and are then no longer traceable" [19]. In well-developed people, the pelvic viscera collectively are separated from these parietal condensations (except anteriorly in the region of the pelvic floor aperture) by fat-laden connective tissue; and only in thin and ill-nourished individuals, such as usually form the subjects for dissection, are membranous strands to be found.

Some of these thickenings, however, have been described as ligaments—or as true ligaments—of the viscera. Thus, the anterior and the lateral true ligaments of the bladder are such thickenings—the former passing directly forwards beneath the bladder, the latter antero-laterally. Mackenrodt's ligaments are similar thickenings, passing outwards from the supra-vaginal cervix and adjoining part of the vagina to the lateral or postero-lateral pelvic walls (white line, ischial spine). Thickenings are also present posteriorly, and pass backwards and upwards from the cervix by the sides of and encircling the gut, in the depths of the utero-sacral folds, towards the sacrum (3rd and 4th foramina) and great sacro-sciatic notches (utero-sacral ligaments). And because of the supposed important physiological function assigned to these thickenings, considerable attention has been paid to them. Thus, Ovenden has described with particular care the attachments of Mackenrodt's ligament, marking out indeed a particular area on the lateral aspect of the cervix uteri for its origin [77]. Others have laid stress on the utero-sacral ligaments; others, again, have

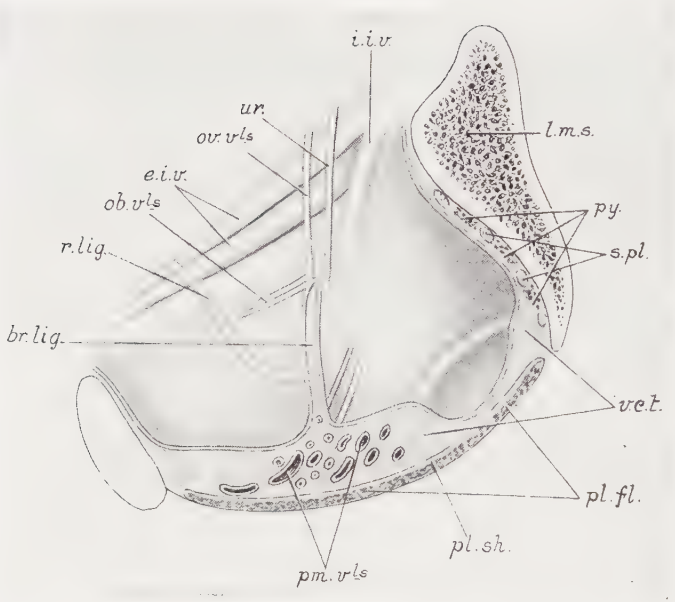

FIG. 22.—AN OBLIQUE VERTICAL SECTION OF THE PELVIC CAVITY PASSING THROUGH THE OS PUBIS AND THE LATERAL MASS OF THE SACRUM TO SHOW THE RELATION OF THE VISCERAL CONNECTIVE TISSUE WITH ITS EMBEDDED VESSELS TO THE PELVIC FLOOR.

(Diagrammatic: according to Cameron.)

Note the thickness with which the pelvic floor musculature is represented, and the gentle curve it is shown to form from behind forwards.

i.i.v. = internal iliac vessels.
ur. = ureter.
ov.vls. = ovarian vessels.
e.i.v. = external iliac vessels.
ob.vls. = obturator vessels.
r.lig. = round ligament.
br.lig. = broad ligament.
l.m.s. = lateral mass of sacrum.
py. = pyriformis muscle.
s.pl. = sacral plexus of nerves.
v.c.t. = visceral connective tissue.
pl.fl. = pelvic floor.
pl.sh. = pelvic sheath.
p.m.vls. = parametric vessels.

[*To face page* 263

directed attention to the pubo-vesical ligaments. Martin has gathered all together and referred to the several thickenings collectively, which as we see radiate outwards in all directions from the cervix towards the pelvic walls, as the "retinaculum uteri." According to this author, with the other intervening parts of the connective tissue, they form a sort of fascial diaphragm or network let into the pelvis, on which the pelvic viscera rest and by which they are supported [61].

It is, however, to be noticed that these thickenings abut upon the pelvic floor, or they are closely related to it. The anterior true ligaments of the bladder (or the pubo-vesical ligaments) are but the condensed peripheral portions of the visceral connective tissue related to the pubo-coccygei. The lateral true ligament is similar tissue more obliquely placed. According to Cameron, Mackenrodt's ligament . . . "may be recognised as the perivascular tissue plus the pelvic sheath of the levator ani and coccygeus to which it is attached below." Mackenrodt's ligament therefore abuts upon the pelvic floor (Fig. 22). And the utero-sacral ligaments, if they do not rest on the pelvic floor and posterior pelvic walls, are not far removed from these parts. There is no interval between these thickenings and the pelvic floor; or, if there is an interval as perhaps there is in well-developed people, the interval is occupied by incompressible material (fat-laden connective tissue). The pelvic floor is obviously in close relationship with these thickenings, as it is to the basal parts of the viscera themselves, from which or by which they pass. These thickenings are certainly deep down in the pelvis, and may be regarded for the most part as condensations of the parietal portion of the visceral connective tissue.

It is moreover to be noticed that these thickenings, which are most marked centrally—that is, where the

cervix happens to be—all disperse and fray out peripherally. Martin expressly describes this dispersion, and clearly depicts it. "In the neighbourhood of the uterus," he says, "the white glistening strong fibres of which the thickenings are composed form an almost homogeneous plate; but towards the periphery they radiate out in a fan-like way, leaving smaller and larger meshes between filled with yellow glistening droplets of fat. Towards the pelvic walls, the thick strands disperse and lose themselves gradually in delicate fibres, which then insensibly cease in the loose cellular tissue. Individual outrunners of these can clearly be followed to the fascia on the pelvic walls. This disposition of these thickenings corresponds with the aggregation of fat-masses found in the subperitoneal tissue peripherally; whilst on the contrary, in the middle about the cervix, even isolated fat droplets do not occur" [61]. Derry's account is therefore confirmed. According to Halban and Tandler, the dispersion is such that peripherally the thickenings are scarcely to be found. But Martin thinks by this dispersion the strain of supporting the viscera is diffused around the pelvic walls, and sees in it evidence in support of his conception that this retinaculum uteri suspends the uterus in position.

These thickenings, however, are not present in the new-born. "If one examines the loose pelvic cellular tissue in the new-born," says Martin, "it appears as a homogeneous mass." Only in individual cases was he able by a special process to find delicate white fibres or strands. Thus . . . 'the well-developed thickenings found in the adult must be regarded as the product of extra-uterine life" (Martin). The occurrence of these thickenings, we have seen, is explained by ontogeny. The gradual rise of pressure in the pelvis and especially the gradually increasing excursions, varied and frequently repeated during activity, by thrusting

the viscera into the pelvis whereby this distends, naturally affect the visceral connective tissue in which the viscera are embedded, causing variations of tension within it to arise. Since centrally situated organs must be most affected by such pressure waves and their resulting displacement be greater than that of peripheral parts, it is easy to understand that the lines of stress arising in the visceral connective tissue must be most marked centrally, and must radiate outwards towards the periphery and affect the visceral connective tissue most medianly and next in these directions.

That the thickenings arise in this way is evident from a consideration of the mechanism of hypertrophy of tissues in general, and of the cause of the perivascular thickenings and of the development of the connective tissue investing muscles into sheaths, of which we shall have something to say. In a similar way, the condensation of the parietal portion of the visceral connective tissue abutting upon the pelvic floor, especially in the region of its aperture (i.e. on the upper surfaces of the pubo-coccygei) and in the region of the great sacro-sciatic foramina, is explained. For in both these parts some yielding to the pressure variations is possible and in health occurs, and thus similar lines of stress must arise and result in a condensation of the affected connective tissue.

3. The Perivascular Thickenings and their Definition

The other thickenings of the visceral connective tissue are found about the blood vessels and the associated nerves which run with the blood vessels. Such thickenings exist wherever vessels traverse this connective tissue: they are not peculiar to the vessels passing inwards from the pelvic walls to the viscera, but are marked about the

main trunks applied to the pelvic walls (internal iliac vessels) and about those which run down along the pelvic brim to enter the thigh (external iliac vessels). Those surrounding the visceral vessels are continuous proximally with the parietal condensation in the region of the great sacro-sciatic notch and with the condensation about the internal iliac vessels from which the visceral vessels take origin—from which condensations these vascular thickenings may be said to arise. Distally, they pass with the vessels to the viscera—to the rectum, to the bladder, and to the uterus (cervix): and in doing so, diverge as the vessels they encompass separate.

Three such thickenings, then, may be described. A posterior, about the middle hæmorrhoidal vessels; an anterior surrounding the superior vesical vessels—the artery of which in its beginning represents the remaining patent part of the fœtal hypogastric, and the branches of which supply the bladder laterally and its vertex; and a third about the uterine vessels. With the pelvis in a position such as obtains when the individual is standing erect, the posterior thickening passes inwards, downwards and backwards, behind the peritoneum to reach the rectum: the anterior passes forwards and downwards and somewhat inwards along the lateral pelvic wall to reach the bladder where this organ comes into relation with the lateral pelvic wall: the third thickening courses obliquely downwards and inwards, across the pelvic space, in between these other two. Reaching the cervix, to which its main artery is directed, this thickening becomes continuous with the condensed connective tissue in this region; and its contained artery divides into branches to supply surrounding parts. Though the chief destination of the uterine is the uterus, this vessel often gives origin to the vaginal artery, which in the female takes the place of the inferior vesical of the male and

supplies the vagina and bladder base. At other times, the vaginal artery arises directly from the internal iliac, and then runs with the uterine in the base of the broad ligament in the same " perivascular thickening."

With these arteries run various veins, or plexuses of veins, abundant in the pelvis. They are necessarily embedded in the visceral connective tissue; and this, condensed about them as about the arteries, forms for them more or less inconspicuous fibrous sheaths. These condensations comprise part of the perivascular thickenings referred to. But, as we shall see, much more than mere thickenings of the connective tissue immediately surrounding blood vessels is included by many authors in these perivascular thickenings, and especially is this so by those who believe that the perivascular thickenings suspend (or support) the pelvic viscera.

Two reasons especially have been advanced for this view: (1) the demonstration during vaginal hysterectomy, that before division of the " parametrium " the uterus cannot be greatly displaced; and (2) the observation that these perivascular thickenings possess considerable density, and in the dead obviously bind the vessels to surrounding parts (as in the limbs) and in the pelvis form a link—apparently a suspending one—between the viscera and the pelvic walls. Fothergill, who is mainly responsible for this idea, says that until the parametrium is divided on either side during the operation mentioned, the uterus . . . " is, for practical purposes, as completely supported as before the incision "—i.e. as before the operation was begun; and that therefore . . . " the vessels and other structures with their sheaths or fascial coverings, which lie on either side of the uterus, below the broad ligaments and above the lateral fornices, are the structures which support the uterus " [27]. Similarly, he believes that the bladder is

kept up in place by the perivascular sheaths of the vessels passing to it, and that the rectum is supported in the same way.

In these vascular structures, which are bi-laterally and more or less symmetrically placed, and which present as successive pairs about the viscera, we recognise but parts or segments of the "parametric swing-bridge" already referred to. By them the respective viscera are supposed to be suspended. When the anterior segment becomes "relaxed" the bladder comes down; when the middle is relaxed, the uterus comes down; when the posterior is similarly affected, it is the rectum which prolapses. The lesion, we are told, which "permits" cystocele—i.e. prolapse of the bladder without that of the uterus—"is relaxation of the paravesical and paravaginal vascular sheaths, the parametric connective tissue remaining normal" [28].

Apart from the impossibility of formulating a pathological basis of so particular a primary lesion, as that of the paravesical and paravaginal vascular sheaths stated to explain cystocele, a lesion which as we have seen is secondary not primary to the displacement, many other considerations indicate the speciousness of this hypothesis. We have been at pains to show that under ordinary conditions of life, the position of the uterus and the limitation of its displacement depend on other factors than the existence of the connective tissue attachments of the uterus; and that the position of the bladder is maintained and affected in a precisely similar way. We have further shown that the conditions prevailing during the operation of vaginal hysterectomy are so different from those obtaining at other times as to be incomparable with them and not analogous; and that although the vessels and connective tissue attachments of the uterus prevent great displacement of the uterus downwards by traction, this does not show that

these structures maintain the uterus in its position in the normal and under normal conditions. Thus, the observation that these structures limit displacement of the uterus during the operation referred to, so far from being a reason for believing they do so in the normal, is wanting in this respect.

Moreover, the disposition of these perivascular thickenings, which is such as to support this hypothesis, can be otherwise explained. These thickenings are necessarily placed laterally in the pelvis on either side of the pelvic viscera; for the parent trunk, of which their contained vessels are branches, is located postero-laterally in the pelvis, not far from the pelvic brim, whilst the pelvic viscera are not only medianly placed, but occupy most of the room in the pelvic depths. And these thickenings necessarily diverge as they pass to the viscera, just as the strings uniting a collection of toy balloons diverge from the place where they are held or tied together—whether the strings support them or no; for the vessels about which these thickenings are formed, are grouped together at their origins, arising in close proximity to each other, whilst distally they are separated by reason of the bulk of the viscera to which they are related. And, in the erect posture, the thickenings necessarily pass downwards: for in the course of ontogeny, the pelvic viscera have been caused to descend, and the vessels supplying them have naturally adapted themselves to the slowly occurring but progressive visceral change. Thus, the positions and downward course of the vessels and their thickenings, which at first sight favour the view that these structures suspend the pelvic viscera, is seen at once—from the changes they themselves have undergone—to be a reason for discrediting such an opinion.

Of significance, too, is the discovery that much more than

the mere condensations of the connective tissue immediately surrounding the vessels is included in these perivascular thickenings. According to the author and some supporters of this hypothesis, the perivascular tissue in the pelvis is different in kind and in extent from that found elsewhere—e.g. in the limbs. Besides the mere fibrous investments of vessels, it embraces other thickenings of the visceral connective tissue we have mentioned—parietal condensations —so-called visceral ligaments. Especially is this so in the case of Mackenrodt's ligaments, which in the past have received so much attention: the integrate existence of these, strange to say, is denied by these authors.

Thus, Fothergill says Mackenrodt's ligaments are firm connective tissue sheaths derived from the pelvic fascia, interspersed with plain muscle, which accompany the uterine arteries [27]. Moritz bluntly states that Mackenrodt's ligaments are artefacts and that Ovenden's picture of their attachment to the cervix is more imaginary than real. "It is impossible," he says, "to demonstrate or define them as separate entities from the remaining parametric tissue. . . . The whole structure is nothing but an inseparable continuation of parametrium which surrounds and fixes the cervix" [65]. According to him, the structure described by Mackenrodt is in reality the vascular pedicle of the uterus. It is . . . "simply the pelvic areolar tissue strengthened by perivascular sheaths, the directions of which are of course those of the blood vessels which they invest" [65]. Similarly, the utero-sacral ligaments are but the continuations of the rectal stalks described by Elliot Smith; they consist of nothing but the nerve supply to the uterus and some smooth muscle, common to all the parametrium, dissected free from its true relations [65].

Such a radical change of view of the structure of the pelvic visceral connective tissue does not furnish the *coup de grâce* to the connective tissue hypothesis; but it does

render the idea that the pelvic viscera are suspended by the condensations about the vessels—the vascular sheaths—much less acceptable or even absurd. The perivascular connective tissue in the pelvis must obviously be considered to be the connective tissue strictly related to the vessels and in close proximity to them; and is to be defined by homology, by a comparison with the similar condensations elsewhere. To the density of this tissue and its significance we shall return. Whatever this may be, it is clear the inclusion with it of other thickenings of the visceral connective tissue in the work of the visceral support, robs the vascular condensations of much of their supposed importance.

But such an attitude is not adopted even by all those who accept Fothergill's hypothesis. Thus, Cameron, although following Fothergill as much as possible, includes other thickenings than those about the vessels in the mechanism of the visceral support. He regards . . . " the perivascular fascia, *plus* the pelvic sheaths of the levator ani and coccygei muscles, as the most important support of the pelvic viscera. . . ." And the pelvic sheaths of the levator ani and coccygei are, for him, not perivascular in nature. He is of the opinion, or he inclines to the opinion, that they represent those parts of the levator ani and coccygei muscles which in the course of phylogenetic evolution have suffered regression [14]. This, in parenthesis, we may observe is a very remarkable observation; for in his paper, he includes this pelvic sheath as part of the visceral connective tissue or fascia, definitely stating it comprises part of Mackenrodt's ligament. Thus, at the same time, this pelvic sheath belongs both to the parietal and to the visceral connective tissue—a fine example of a contradiction.

Other writers are more definite and clearly oppose Fothergill. Thus, Herbert Spencer opposes the idea that

Mackenrodt's ligament consists of perivascular tissue. The "ligamentum transversale colli," he says, lies at a lower level than the uterine artery and is very dense in structure: any one who has noticed this whilst performing total abdominal hysterectomy will find it difficult to accept the statement that it is the sheaths of the vessels which keep the uterus in place. And, how can the bladder be kept up by the sheaths of the vessels supplying it which are small in number and size? [94]. Victor Bonney, referring to the utero-sacral and Mackenrodt's ligaments, says, . . . "these radiating masses of fibro-cellular tissue are very definite structures, and play an important part in maintaining the position of the vaginal vault and cervix, (as) is obvious to all who have performed the modern radical operations for carcinoma of the cervix." He clearly considers them distinct from and not to be confused with the perivascular thickenings; for these latter, he says (according to him, apparently, of secondary importance), are to be included with the ligaments mentioned in the structures forming "The Middle Supporting Group" of the Sustenacular Apparatus [12].

Many other gynæcologists however, who believe the "perivascular tissues" play a part—some even believe, an important part—in the support of the uterus are somewhat indefinite about it. Eden and Lockyer, for instance, lay considerable stress on the perivascular tissue from this point of view [26]. But these authors, like some others, include in the uterine support (except the broad and round ligaments, which have been universally rejected) all other possible structures, appearing unable to fix their minds definitely on one. Moreover, it is not clear they have defined for themselves the meaning of the term "perivascular tissue"—presumably, we are to understand by it the dense tissue, not merely forming the fibrous investments of the vessels, merely their sheaths;

but that found in the neighbourhood of the vessels, more or less related to them—as for instance is the dense tissue known as Mackenrodt's ligament. Stevens is in like case. Although insisting on the importance of the pelvic floor and rejecting Fothergill's conception, he says that the connective tissue at the base of the broad ligament around the uterine artery—which is synonymous for him with Mackenrodt's ligament—and the connective tissue about the vessels supplying the vagina and bladder, cannot be overlooked, and must be regarded as at least a contributory factor [96].

There can be no doubt that, according to the majority of authors, the parietal condensations and the perivascular thickenings are distinct from each other. The latter, naturally, are less superficially placed than the former. Whilst the so-called visceral ligaments, collectively forming the "retinaculum uteri," are in close relationship with the floor and walls of the pelvis, radiating outwards and upwards from the cervix—in front, at the sides and behind; the vessels with their fibrous investments are more deeply placed, for they traverse the connective tissue to reach the viscera, radiating downwards and inwards to the several organs—anteriorly, medianly and posteriorly—from the postero-lateral pelvic walls above. But whilst this is so, there is also no doubt that these two distinct kinds of thickenings have been confused with each other. Thickenings have been called perivascular even though not in the immediate vicinity of vessels and even though containing non-striped muscle. According to Moritz, the thickenings at the sides of the uterus, although composed of fibres which take for their direction the branches of the uterine arteries, yet pervade adjacent parts of the visceral connective tissue—in one direction, they run into the uterus; but in the other, spread out . . . "forming sheaths for

the obturator internus, part of the pyriformis and the pubo-coccygeus " [67].

Moreover, an essential ingredient of them is smooth muscle (Moritz). In parts, this is said to be conspicuous, even predominant: the lower uterine segment, says Moritz, is fixed and held in position by tier upon tier of dense connective tissue and by masses of smooth muscle. But thickenings with a content of smooth muscle are much more likely to be visceral than vascular in origin. We know quite well that the musculature of the uterus gives rise to strands of smooth muscle cells which pass outwards and disperse in the visceral connective tissue. On the other hand, the condensations about blood vessels elsewhere (e.g. in the limbs) are essentially fibrous—as far as is known they do not contain non-striped muscle cells. All the so-called visceral ligaments—the pubo-vesical, Mackenrodt's, and the utero-sacral—contain smooth muscle. Some of these, or parts of them, obviously have been included with the specific thickenings about the pelvic blood vessels under the name " perivascular."

One reason for this seems to be that the vascular and visceral condensations are more or less closely related to each other. Especially is this so in the case of the vascular condensation about the uterine vessels; for this runs inwards to the cervix, parallel with, but just above—on the summit of—Mackenrodt's ligament, or at least with the posterior part of this ligament—a structure which appears co-extensive and identical with the visceral ligament of Paterson. And it is this perivascular thickening to which attention has been more or less exclusively directed; as we have urged the perivascular thickenings about the vesical vessels and about the middle hæmorrhoidal have been more or less neglected. But much more significant than this close relationship is the well recognised fact that all the thickenings—the perivascular as well as

the parietal condensations—are indefinable and pass insensibly into the surrounding looser connective tissue.

Both, moreover, are highly variable, being of different extent and magnitude in different women. Even in the same individual, as we have seen, the pelvic visceral connective tissue undergoes great changes during the life history of that individual. Especially is this so in pregnancy, in which state an atrophic process sets in. In this condition, the enlarged lower segment of the uterus comes to encroach upon the space occupied by the normally fat-laden connective tissue in the pelvis; it does so, not by displacing this connective tissue to other regions (as into the abdomen), but by causing it in part to disappear—the fat undergoing autolysis and its particles being removed by the blood or lymph stream. And the thickenings of this connective tissue also seem to suffer—their condition in pregnancy does not seem to have been investigated by anatomists; at any rate, they do not present the same difficulties to the surgeon engaged in removing the uterus when pregnant, as it is stated they do in the non-pregnant woman. Apparently, they are not so dense, or not at all dense; they are easily cut through, the pregnant uterus being easily removable. We have already dwelt on the succulence and looseness of the pelvic visceral connective tissue in pregnancy; and this condition is well known. We may well ask: How can this tissue, with whatsoever thickenings it may then contain, support or suspend the pelvic viscera in pregnancy? If it cannot be supposed to do so in pregnancy, how can it be believed it can do so in non-pregnant women in virtue of the thickenings it then possesses?

All those who believe the perivascular thickenings, with or without the other thickenings, play either the principal or a subordinate rôle in the statics of the female pelvic

viscera, must believe these thickenings act by suspending the organs. Fothergill has depicted the mechanism as he sees it by the illustration of the parametric swing bridge: and Moritz leaves us in no doubt as to his meaning. He says that if numerous elastic bands are placed around the cervical segment of a model uterus, and have the same tension as in a pelvis in the erect posture, the model takes up a position of anteversion. And that . . . "Different areas of the elastic strands being loosed causes the model to assume different displacements" [67].

Such bands resemble much more the retinaculum uteri than the vascular condensations; but whether we consider the action of either separately or of both together, such conception is plainly inapplicable physiologically. Quite apart from the difficulty of discovering the tension of such bands in the living and of reproducing them in a model, it is plain the mechanical necessities for the maintenance of the position of the uterus in the pelvis—a part of the body, that is, the position of which itself in the living is constantly changing—cannot be met alone either by bands radiating outwards in all directions from the cervix; or, on the other hand, by bands only related to the uterine arteries, passing only from the cervix to the postero-lateral region of the pelvis, possessing only one general direction. The retinaculum uteri presents the more reasonable mechanism, but even this fails. How, for instance, can a uterus, displaced backwards and upwards by a distended bladder, be caused to assume again a position of anteversion in a woman in the dorsal decubitus, when the bladder is emptied by catheter by either of these mechanisms, or by both acting together? If they act at all, they can only do so on the cervix; but no one will believe that traction on the cervix by such means alone could cause in such a case the replacement of the fundus uteri anteriorly. It is plain some other factor must come into play, such as the volume and

incompressible nature of the parts surrounding the uterus, and the pressure with which they are pressed against the several parts of the organ. But the moment one brings these pressure conditions into consideration, the necessity for a suspension by bands disappears.

Such conditions, however, do not come into the conception of Moritz. He believes the existence of non-striped muscle in the parametrium explains everything. On this tissue, as we have seen, he lays great stress. Though more marked in certain parts, the non-striped muscle more or less pervades the parametrium: if perivascular in nature, it is not limited to the immediate vicinity of the pelvic visceral vessels. Physiologically, it is highly important. In virtue of it, the thickenings of the visceral connective tissue are more than "elastic:" they possess a "definite tonus," and are "actively contractile." Indeed, Moritz attributes such qualities to the pelvic visceral connective tissue itself as a whole. In virtue of the non-striped muscle it contains and the qualities which this gives it, the pelvic visceral connective tissue is "ever variable in dimensions (sic) and thus able to arrange itself for the varying conditions to which the pelvic viscera are liable" [67].

In support of his contention, Moritz adduces biological evidence, which is interesting and perhaps important. For, besides its ontogenetic change, the pelvic visceral connective tissue, he says, has undergone a phylogenetic evolution—an evolution, comparable with, but presumably surpassing in significance that which the pelvic floor itself has undergone. "The retro-peritoneal pelvic connective tissues have evolved in man," he says, "simultaneously, with other modifications, on the assumption of the erect attitude." Whilst an examination of these tissues in lower animals—such as cats, rabbits and hedgehogs—shows that in them these tissues are devoid of smooth muscle fibres, consisting only of a loose reticular tissue with large spaces filled with fat,

the higher the vertebrate type the more condensed does this tissue become and the more smooth muscle does it come to contain. "The tailed monkeys possess a connective tissue which is still poor in smooth muscle and supporting elements, but these are present in larger amounts than in the connective tissue of quadrupeds. . . . (But) the pelvic connective tissues of the anthropoid apes are exceedingly rich in smooth muscle and supporting tissue. . . . It is therefore proved that the higher we go in the scale the richer this tissue becomes in smooth muscle and the better it is thus adapted to the mechanical duty of keeping the pelvic viscera in position" [67].

This is the evidence on which Moritz bases his concluding remark that the visceral connective tissue has obviously been evolved and is eminently adapted for the function of the visceral support. It is not the only evidence he gives for this opinion; but it is the only evidence necessary to consider.

4. The Perivascular Thickenings and their Significance

Without attempting, for the moment, to refute such an idea—of impossible application to the living body—let us consider the reason for the existence of the thickenings about the pelvic visceral vessels—the fibrous investments, which we have noticed are not peculiar to these vessels. The fact that this is so, that other vessels are similarly invested, is highly important, shedding light on the ætiology and purpose (if any) of the perivascular sheaths in the pelvis. The existence of sheaths for arteries and veins, we know, is characteristic of all such vessels: fibrous condensations of the surrounding connective tissue exist throughout the breadth and extent of the vascular system, except (under normal conditions) in the region of the

permanent capillary beds. And these condensations arise with the development of such vessels; . . . as a result of their development: the perivascular sheaths are a by-product of vascular development!

They certainly arise with this development. The vessels that enter the limbs, for instance—as all other vessels (even the aorta)—begin as capillaries or as groups of capillaries, and have at their beginning no perivascular sheaths. The connective tissue about them is highly cellular, and consists of a process from the mesodermal matrix in which the viscera themselves and the vascular mechanism are developing. But with *the passage of time* —that is, as development proceeds and capillaries become changed into arteries and veins—the connective tissue cells about them undergo corresponding changes. The connective tissue cells immediately abutting on the endothelial walls, become polyhedral or cubical, and by their further metamorphosis form the specific structure of the developed vessel wall. But the connective tissue cells outside those taking on a specific function and structure, give rise to extra-cellular fibres, themselves ultimately disappearing. They come to form the adventitious coats of arteries and the corresponding parts of veins; and sheaths for the vessels collectively, within which nerves—because of their proximity—are sometimes included or embedded (as, for example, happens in the case of the sheath about the carotid vessels).

What is the cause of this change? Clearly, the universal thickening of the connective tissue about the larger vessels can only have a universal cause, a cause which affects—except permanent capillaries—the whole vascular system. Such a cause is to be found in the gradual rise of blood pressure within the developing vessels, which becomes, as growth proceeds, gradually greater and greater than the pressure of the parts immediately surrounding them.

This rise of pressure is due to the demands for an increasing blood supply to the parts beyond: its direct cause is an increased activity of the heart. At first it affects capillaries developing into arteries; subsequently, and to a less degree, those developing into veins. Though the rise is continuous, it is not regular: variations occur as the activities of the parts supplied wax and wane. Besides these, more marked variations begin to show themselves, especially (perhaps only) in the arterial capillaries, as the pressure waves due to the heart beat reach them. The immediate effect of such change is change in volume of the vessel concerned, with a consequent and corresponding disturbance of the surrounding cellular matrix. The cells of this matrix abutting upon the endothelial wall of the developing capillary take upon themselves the function of opposing the excess of pressure (as compared with the pressure of the parts outside) now appearing, becoming permanent, and with development increasing within the vessel; those immediately outside these, becoming subject to increasing variations of tension, like connective tissue cells elsewhere, react and condense.

Many considerations show this is so. In the first place, permanent capillaries only remain as capillaries because under normal conditions the blood pressure within them is always, or practically always, the same as the cellular pressure immediately outside them—the pressure wave from the heart beat does not reach them; and did great differences obtain, the capillaries would either burst or collapse. These thin-walled vessels, consisting only of flattened endothelial plates, are incapable of sustaining a pressure within them greater than is the pressure without: that is why they are such efficient functional structures, allowing the easy interchange of products (by osmosis) between the tissues and the blood. But when the pressure within

capillaries becomes gradually greater and greater, as happens pathologically, as (for instance) in cases of heart disease and of emphysema, and normally during development, not only the capillaries but the tissues immediately about the capillaries, alter—a fibrosis ensues; and the interchange between the tissues and the blood within these vessels becomes impaired or abolished.

And, in the next place, although the walls of arteries and veins are of a specific structure and can sustain considerable pressures within them, they do not prevent the surrounding connective tissue from being subject to variations, not only of pressure but of tension. Obviously, such must occur with the pulsation of arteries—their expansion due to the heart beat; and in a lesser degree with the changes in volume veins undergo. With the enlargement of vessels, the result of increased pressure within them, the surrounding parts are more pressed on; and if displaceable are displaced: with their shrinkage, due to a fall of pressure within, the parts outside are pressed on less; and if previously displaced return to their former positions. Whilst the connective tissue matrix in which the vessels lie, is subjected to such changes of pressure; since it completely surrounds the vessels, their variation in volume necessarily must cause as well variations of tension within it, especially in that part abutting on the vessel which as the vessel expands must embrace it more tightly.

That the connective tissue is affected by such variations of tension is shown by the differences found in the condensations about different vessels. The condensation about arteries is greater than that about the corresponding veins, because the pressure in arteries is greater than in their veins, and much greater variations of pressure occur. Arteries, for this reason, expand more markedly than do veins; they pulsate, and thus the connective tissue about

them undergoes much greater variations of tension than does the connective tissue about their veins. Similarly, we find the condensation varies with the size of the vessels: for the larger the vessel not only is the pressure within it higher, but the variations of pressure are greater, and consequently so much the greater are the variations of tension to which the surrounding connective tissue is subjected. Thus is explained the fact that the condensation about the external iliac vessels is more marked, is denser and more obvious than is that about the uterine vessels. Pathologically, we see the cause at play in the growth of the adventitia and surrounding connective tissue in cases of aneurysm; in which, owing to the failure of the specific structure of the vessel's wall, the surrounding connective tissue is more greatly affected than under normal conditions.

Nor is this condensation peculiar to the connective tissue about vessels; that in which muscles are enveloped undergoes similar and analogous changes. In the embryonic condition this also is cellular [53]; but with growth it undergoes changes forming sheaths for the muscles. And the greater the activity of the muscle, the greater is the fibrotic change—peri-muscular sheaths are more marked the greater the development of the muscle. In weak and delicate individuals they are inconspicuous; in robust active people, they are well developed. Here, too, it is plain, the changes of tension to which the connective tissue envelopes are exposed during the activity of the muscle are the cause of this condensation. Contraction of a muscle, resulting in its shortening, will necessarily cause its connective tissue sheath to be bulged as the muscle bulges, a tension in the fibres of the sheath occurring circumferentially to the length of the muscle: on the other hand, elongation of the muscle, due to the contraction of antagonists, will induce a tension in the fibres of its sheath

in a direction parallel with the muscular fibres. Such tension will vary with the activity of the muscle—not only with the degree of its contraction and relaxation ; but also with the extent of its shortening and elongation (conditions, which, respectively, do not necessarily occur together).

The existence of the perivascular thickenings being explained by the expansion of vessels and the variations of tension to which the connective tissue in which they are embedded is subjected—both during the ontogenetic development of the vascular system and in the adult during those vascular changes associated with hypertrophy of parts or organs—it is not surprising that the arteries and veins in the pelvis should possess such sheaths. But if, for a moment, we would question why they are there, let us first ask ourselves the purpose of the similar structures about blood vessels elsewhere—about those, for instance, supplying the limbs and head and neck, which are encompassed by sheaths so conspicuous and apparently so strong. It is plainly absurd—is it not ?—to suppose these sustain in position the parts to which they are immediately related or the parts beyond to which the vessels go. What structure does the sheath of the external iliac vessels support ? What, the carotid sheath ? . . . Nor can it be supposed they maintain the vessels themselves in place : such is as absurd as to think the sheaths of muscles maintain muscles in position. The vessel walls are stronger than the perivascular sheaths and require no such provision. (They also, we see, have been included in the work of the visceral support.) When the vessels were formed and their paths laid down, their position was obviously determined by the existence (i.e. by the resistance) of surrounding parts.

And, it is these parts—whether muscles or viscera—which, in virtue of the pressure they exert, maintain the

vessels in position. Tumours as they grow easily displace even large vessels, in spite of the perivascular sheaths and the adhesion or continuity of these to other sheaths or condensations. Enlargement of the thyroid, for instance, easily displaces the carotid sheath and all it contains. The growth of pelvic tumours, whether of ovarian or of broad ligament cysts, whether of the uterus as by pregnancy, easily causes changes in the positions and *in the lengths* of the related vessels. On the other hand, when the pressure within vessels becomes greater than normal, the pressure of surrounding parts may be inadequate to maintain them in position. When the pressure in the internal saphena vein becomes abnormally great and the increase is persistent, not only does the vessel enlarge, but it becomes tortuous—i.e. longer. The same is seen in the superficial temporal artery under similar conditions. Nor can more deep-seated tissues, of greater importance and capable of considerable resistance, maintain vessels in place when, for instance, an aneurysm forms. In the presence of such, bones, muscles and viscera are displaced or disappear.

It is plain, the perivascular sheaths do not maintain the vessels in place. And, it is clear, they do not subserve the function of the vessels, or assist them in any way. Fibrosis about capillaries impairs their function; fibrosis about larger vessels does not prevent them enlarging or pulsating, just as it does not prevent them from becoming tortuous or the seat of an aneurysm. Such changes are conditioned by the blood pressure within and the condition of the specific structure of the vessel wall. We know perfectly well, from pathological teaching, that the adventitious coat of arteries, though intimately related to and connected with the vessel wall, forms no part of its essential structure: that in spite of the apparent strength of the adventitia, it is not capable of resisting the blood pressure within. Expansion—pulsation—only occurs when the essential

element yields: only when this becomes diseased can an aneurysm form. It is plain these perivascular thickenings are but passive effects of the variations of pressure to which the connective tissue surrounding blood vessels is subjected—not an active means of sustaining or helping to sustain the pressure within, or of strengthening the vessel wall.

CHAPTER XIII

THE NECESSITY FOR A PELVIC FLOOR

1. The Thoracic Diaphragm and its Meaning

With such an opinion, we come back to the perivascular sheaths in the pelvis, and to the idea that these, with their vessels, suspend the pelvic viscera. We cannot help being surprised at this idea. The more we consider it, the more impossible does it become. When we remember how easily arteries elongate, in spite of their muscular coats, and even though in some cases their walls contain such tissue arranged longitudinally, and in spite of their perivascular sheaths, we cannot believe them capable of suspending the viscera. Indeed, we cannot believe the viscera are suspended at all. We cannot believe this, even though anatomists should believe it and promulgate it:—as, for instance, does Wood Jones, who, developing a great idea, says that during evolution, "when the trunk axis becomes more upright, all the viscera tend to sink towards the hind end of the body, and they tend to be suspended from the head end of the cavities rather than from the dorsal aspect; . . ."—for instance, that "The liver is suspended more strongly from the lower surface of the diaphragm, and the intestines obtain attachments which sling them from the upper part of the cavity, as well as from its dorsal wall" [46].

Anatomists come to this opinion from the conditions found in the dead—the existence of vascular pedicles, dense and strong, and of other ligamentous bands passing

to the viscera, which, however, hardly suffice to explain a liver suspension. But a consideration of the dynamics in the living would cause such anatomists as hold this opinion to change it * ; for the conditions found in the living are incompatible with it. We cannot believe the heavy liver is suspended from the diaphragm ; either by its direct attachment to the diaphragm posteriorly (i.e. by a more or less extensive area uncovered by peritoneum) ; or by means of the coronary, lateral and falciform ligaments (the last surnamed suspensory) ; or by the large hepatic veins with their sheaths ; or by all these collectively. Indeed, these structures are not needed for the liver suspension: capillary adhesion between the liver and diaphragm, even in the region of the peritoneal invaginations, would suffice to explain it. But we reject this idea for several reasons.

When we look at the position and movements of the diaphragm in the living man, which we can readily do by means of the X-rays, we see the right cupola of the diaphragm pushed up into the chest to a much greater extent than is the left cupola. If the liver, which weighs more than the stomach even after a meal, were suspended by the diaphragm, the reverse should be the case. For, if the epigastric viscera are suspended, they cannot be suspended in virtue of the diaphragm *per se*—the form and structure of the diaphragm prohibit such an idea. If suspended, their suspension must be due to something beyond—above—the diaphragm ; it must be attributed either to the mediastinal fascia or to the negative pressure in the pleural sacs or chest—i.e. to the elasticity of the lungs.

But neither of these factors explains the peculiarities referred to. Although the mediastinal fascia has connections above with the cervical fascia and so with the

* Happily, this opinion is not universal amongst anatomists.

base of the skull and the upper vertebral column, it only reaches the diaphragm by means of the pericardium, which is attached to the central tendon. Even though it should be thought that this fascia suspends the heart and central part of the diaphragm (which it is as incapable of doing as are the similar structures in the abdomen of suspending the abdominal viscera),* it clearly cannot be considered the cause of the positions of the more lateral parts of the diaphragm which are more highly placed than is the central tendon. If we suppose the negative pressure in the pleural sacs is the cause of the positions of the right and left cupolæ of the diaphragm respectively, to explain the higher position of the right side of the diaphragm, we must suppose the negative pressure on the right side, in the right pleural sac, is greater (i.e. more negative) than is that on the left side, in the left pleura—that is to say, that the elasticity of the right lung is greater than the elasticity of the left.

Moreover, if the negative pressure in the chest suspends the liver, then the pressure between the liver and the diaphragm must also be negative—as Matthews Duncan thought—for the upper surface of the liver is always in contact with the diaphragm. This must be so, even though the diaphragm by its tonic contraction should

* With descent of the diaphragm, the heart is depressed, following the diaphragm. It is pressed down on the diaphragm, because the difference between the intra-pulmonic pressure (approximately the air-pressure) and the mediastinal pressure (which owing to the elasticity of the lungs is negative) is increased with descent of the diaphragm. Conversely, as the diaphragm reascends the heart also is raised. For the force depressing the heart is diminished as the lungs get smaller, whilst the force which presses up the diaphragm presses up the heart as well.

These changes in the position of the heart with abdominal respiration can easily be seen by the X-rays, and are most interesting. They clearly indicate that the heart is not suspended by the mediastinal fascia.

participate in the production of the negative pressure in the chest. Thus, the elasticity of the lungs has to oppose both the weight of the liver and the tonic contraction of the diaphragm—at least in the erect posture.

But the diaphragm is a highly muscular structure. From its central tendon, muscle fibres pass downwards, peripherally, to the lumbar spine, costal margin and sternum, with various degrees of obliquity. Those behind, forming the crural and arcuate fibres, are steeply placed and are longest. Above, arising from the posterior indentation of the tendon, they pass downwards and backwards, medianly, over the epigastric viscera; then downwards behind them; and then, as the spine advances, they are similarly advanced. Those at the sides are more oblique; but their direction depends on the tone of the diaphragm and state of the thorax. In the relaxed condition, they are more curved—being directed more or less horizontally outwards over the viscera beneath, to pass on approaching the chest-wall, steeply downwards; but when the tone of the diaphragm is good as when the thorax is expanded, they are less curved and more oblique. As we have previously noticed, these lateral fibres do not pass merely outwards and downwards, but they possess a pronounced forward direction as well; they sweep over the underlying viscera in an outward and downward and forward direction. Though they gradually become shorter from behind forwards; laterally they are still of considerable length. The sternal fibres are still more oblique, approaching the horizontal, and, owing to the anterior convexity or tongue of the tendon from which they arise, are the shortest of all.

The conspicuous feature of the diaphragm, clearly, is not its tendon, but the circumferential muscular part. This is peculiar in the direction of its fibres, and in the different lengths of its different parts; and it is of some

thickness. Moreover, it is peculiar in that it varies in its state of development with the volume of the abdominal visceral mass—hypertrophying, for instance, in corpulency, like the flank muscles of the abdominal wall; and in pregnancy, undergoing changes similar to those these other muscles then undergo [47].

It is difficult to believe that such a muscular structure as the diaphragm, which obviously is powerfully contractile as shown by its rôle in cough, has but to overcome the elasticity of the lungs and to produce the negative pressure in the chest—a pressure which, according to Howell, at the end of expiration is but −4·5 mm. Hg., and at the end of inspiration but −7·5 mm. Hg.* [45]. But this difficulty becomes much greater if gravity, acting on the liver and other epigastric viscera, is a factor in the production of that pressure. Yet, if the liver is suspended from the diaphragm, such must be the case. We cannot believe that, under ordinary conditions, the diaphragm is so contracted that the pressure beneath it, between it, and the liver—if it be negative—is without effect or influence on the negative pressure in the chest. Such could only occur with maximal contraction of the diaphragm; but then—the glottis being open—the lungs would be maximally expanded (with a much greater negative pressure in the chest). But, during quiet respiration, the lungs are not maximally expanded—in spite of the glottis being open all the time.

Thus, if the liver be suspended, it would seem that when standing erect, the diaphragm is less necessary than when the individual is lying down. But this is not so: we know the tonic contraction of the diaphragm is greater in the former than in the latter position. Not only is the pressure in the chest more negative when standing than

* The position of the individual to which these figures refer is not stated.

when lying down—a difference, however, which might be attributed to the different effect of gravity in the two cases; but two other differences demonstrate this. If we examine the contour of the diaphragm in the coronal plane in the two positions (by means of a screen and the X-rays), we see that, whilst in the dorsal decubitus the curves formed by the right and left cupolæ and by the central tendon are well marked; when the individual is standing they are less conspicuous—the outline as a whole, from being more or less square in shape, becomes more parabolic. These differences can only be due to a greater tonic contraction of the diaphragm in the erect posture than occurs in the individual when lying down. And, in the second place, the pressure in the stomach (as measured by passing a tube into the organ and connecting it with a manometer) is greater in the erect posture than when in the dorsal decubitus.

Again, the diaphragm in orthograde animals is a better developed muscular sheet than it is in pronograde animals. In spite of the fact that gravity acts favourably in orthograde animals in the production of a negative pressure in the chest than it can possibly do in animals which go about on all fours, the force of the contraction of the diaphragm in the former has, for some reason, to be greater than in the latter type of animal.

These facts are incompatible with the idea that the diaphragm exists merely to overcome the elasticity of the lungs and that the liver is suspended from it. If, in place of this idea, we suppose that the diaphragm not only opposes the elastic recoil of the lungs, but at the same time also opposes the upward thrust of the epigastric viscera—that, instead of suspending the liver, it presses downwards upon it—not only do the difficulties which the muscular nature of the diaphragm raise disappear, but such conception explains the facts we have related. That

the epigastric viscera are thrust upwards is easy to understand, and requires little explanation. The volume of the abdomino-pelvic visceral mass; its incompressible nature; the occlusion of the pelvic outlet; and the presence of the transversales abdominis muscles—collectively—suffice to explain it.

These transversales, too, are interesting muscles. In the region of the costal arch, they pass inwards not only to the outer boundary of the recti muscles, but even beyond —more internally—behind the muscles. To such an extent does this occur that near the summit of the arch, the fibres of one side reach those of the other, the rectal sheath here being represented by transverse muscle fibres (Quain). Thus, the margins of the costal arch, instead of being joined together by an uncontractile aponeurosis, are united by a highly contractile mechanism by which their separation can be actively prevented or resisted and their approach directly determined. And we know quite well that on inspiration, as the diaphragm descends, the costal arch widens and the epigastrium bulges; and that, on expiration, the costal arch narrows as the epigastrium recedes, the epigastric viscera being replaced inwards and upwards (since they cannot pass downwards). This return is caused by the abdominal wall muscles (not by atmospheric pressure); the epigastric viscera, as they are replaced, pushing upwards again the diaphragm above them, which musculature allows and stays—i.e. governs—their return.

That expiration in all mammals is a muscular act is perfectly clear. In us, the effort expended is indeed less than that necessary for inspiration—from which it seems that expiration is but a passive return, a letting go. As a matter of fact, the transverse and other muscles of the abdominal wall play an essential part in expiration. In the new-born child this clearly is so: for even after the

first few inspirations the pressure in the thorax remains positive—does not become negative. The negative pressure in the thorax develops only slowly, not suddenly. It is not demonstrable during the first four days of life; and, according to Hermann, at the eighth day is only −0·4 mm. Hg. [37]. Thus, in the first few days of life the elasticity of the lungs does not come into play for expiration, yet expiration occurs; and clearly it can only be produced by a muscular contraction of the body wall muscles. Moreover, even in the adult, the apparently passive expiration has a limit, beyond which expiration can only be continued by a forcible contraction of the body wall muscles. This is so, in spite of the fact that the lungs are still elastic and a negative pressure still exists in the thorax as it does after the most forcible expiration. Such forcible contraction, especially of the abdominal wall muscles, clearly is necessary because of the resistance of the still contracted diaphragm, for there is no other structure than the diaphragm to occasion it; and it is produced to overcome this resistance of the diaphragm.

Thus we see that the continued contraction of the abdominal wall in ordinary expiration is necessary, not because the lungs are unable to shrink without it, but because the diaphragm opposes the act—opposing it, not violently but gently, for which reason expiration is easy; but opposing it with a force which is greater not less than the force of the elastic recoil of the lungs, so that the pressure in the abdomen may remain ever greater than that in the thorax. Thus, the diaphragm and abdominal wall are antagonists, acting upon each other through the medium of the viscera, which in this way are compressed. Such antagonism explains the muscular nature of the diaphragm, the direction and different lengths of its muscular parts, and the variations found in different individuals or under different conditions. And it explains

the differences found in different mammals. These, which cannot be understood from mere examination of the dead, become clearly intelligible when we consider the habits of the living individuals.

Most interesting in this respect is the diaphragm of such

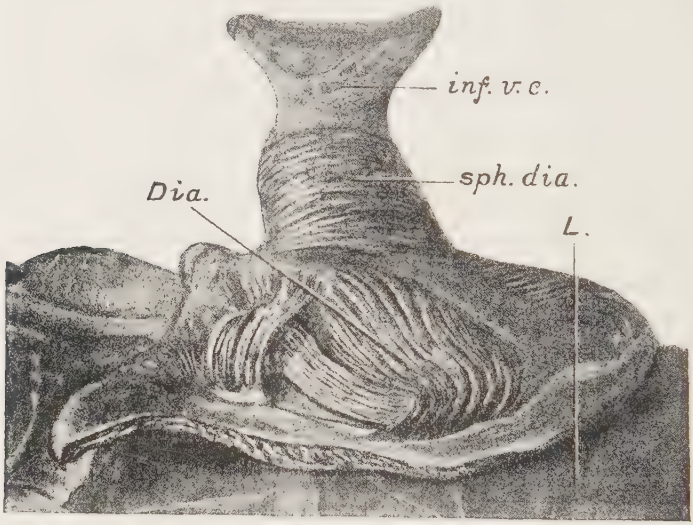

FIG. 23.—THE UPPER MEDIAN SEGMENT OF A VIEW OF A SEAL'S LIVER AND PART OF THE ATTACHED DIAPHRAGM FROM BEHIND.

(From Specimen No. 980 Eb. R.C.S. Museum, ⅔ scale.)

Note the muscularity of the diaphragm and the formation of a sphincter embracing the vena cava inferior by a reflection of its fibres around that vessel in the form of a cuff.

inf. v.c. = inferior vena cava.
sph. dia. = sphincter formed by diaphragm embracing inf. vena cava.
Dia = diaphragm.
L = liver.

aquatic mammals as the walrus and the seal, which because of their aquatic habits are often exposed to pressures to which terrestrial vertebrates are rarely or never subjected. In these animals, it is plain that the effort necessary to maintain the chest expanded is much greater when immersed in water than when on land. The pressure of the

water not only tends to contract the chest, but by pressing the abdomen inwards to thrust up the diaphragm. Not only in these aquatic mammals is the diaphragm much more muscular than it is in their purely terrestrial cousins, to the extent that the central tendon is replaced by muscle; but the vena cava, which in terrestrial forms pierces the tendon, is in these others surrounded by muscle fibres arranged circularly around it, which are even reflected upon the vessel proximally (i.e. on its thoracic side) as a sort of cuff, forming a definite sphincter for it (Fig. 23). Whether this ever actually occludes the vena cava, it is difficult to say: that it governs the calibre of the vessel, we may believe—from the arrangement of the muscle fibres comprising it; that it constricts the vessel to a great extent at certain times (as during immersion) is obvious—since distally to it large venous reservoirs exist in the liver, and are evidently the result of a damming of the blood (Fig. 24). Nothing, perhaps, can show more conspicuously than this the relation of the diaphragm to the pressure conditions in the abdomen and the rôle this musculature plays in regulating the passage of the blood from the abdomen into the thorax.

We can readily understand that, in the erect posture of man, gravity should have some effect on the epigastric viscera, just as in the same position it has an effect on the blood-stream and circulation. But we know that, during health, gravity is ineffective in causing or materially assisting the flow of blood from part to part; for the simple reason that the vascular system and the subsidiary mechanisms concerned in the circulation, by changing their activity as the needs—the activity—of the individual changes, are quite capable of causing the blood to flow adequately to all parts of the body whatever its position, and more to some parts than to others as necessity requires

—for instance, to the brain in the erect posture—the posture of most intense cerebral activity—however great be the opposing or (conversely) the facilitating effect of gravity. Similarly, the apparatus concerned in producing and maintaining a sufficient negative pressure in the chest is in health independent of the effect of gravity. In other words, the mechanism engaged in this is more powerful than is the effect of gravity—we can even hang with our heads dependent and still breathe! And there is no difficulty in believing that the forces at work in the structure of man are capable of producing in him such a controlling mechanism—a mechanism which surpasses in play the effect of external forces. The advantages of such are obvious. If the negative pressure in the chest depended even to a small extent on the effect of gravity, the nicety with which this pressure is regulated would disappear, co-ordination with other body needs would be impossible, and man as a biological experiment would have been unsuccessful.

It is only in pathological conditions in man—as in the normal it is only in respiring non-mammalian vertebrates (i.e. in reptiles and probably in birds)—that the effect of the visceral weight comes into play as a factor influencing the negative pressure in the chest. Just as, under certain conditions, we make use of gravity for facilitating the circulation—as, for instance, in the treatment of collapse; so also, in others, do we make use of it for increasing the negative pressure in the chest. Thus, in certain cases of heart disease, the patient cannot lie down—he feels suffocated; the diaphragm is not strong enough to press the epigastric viscera far enough downwards, or to press upon them with sufficient force: to help himself, the patient sits up (orthopnœa). In this position, under these conditions, the visceral weight comes into play and assists the diaphragm and the patient breathes more easily. But

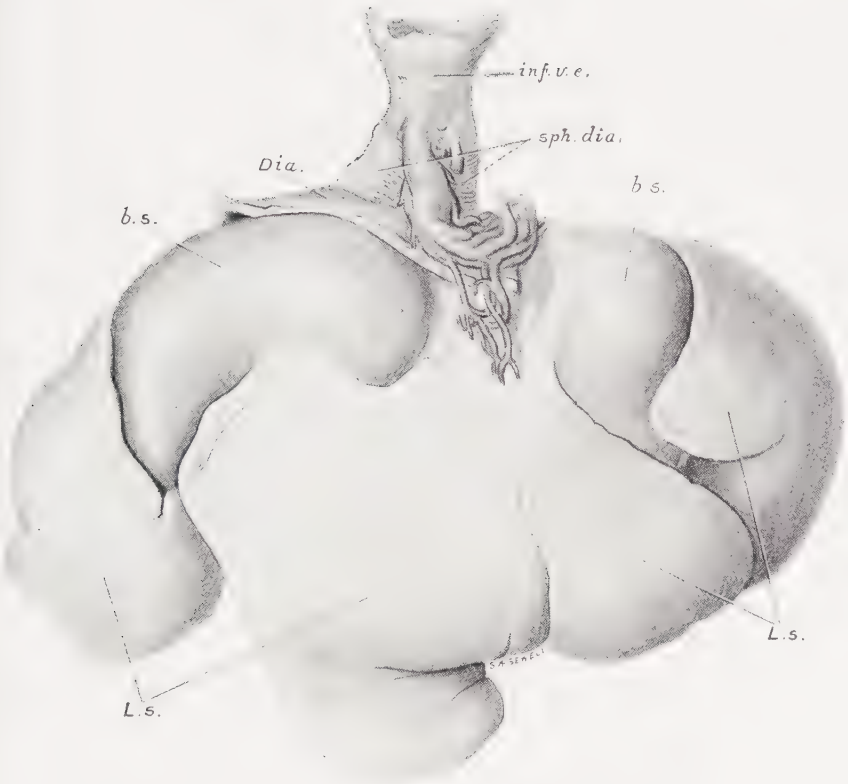

FIG 24.—THE ANTERIOR VIEW OF THE SAME SPECIMEN FROM WHICH FIG. 23 WAS OBTAINED. ONE SEES THE SPHINCTER AROUND THE INFERIOR VENA CAVA FROM IN FRONT, AND THE LARGE VENOUS BLOOD SACS IN THE LIVER.

Considerably reduced.

inf.v.c. = inferior vena cava.
sph. dia. = sphincter muscle around inf. vena cava.
Dia. = diaphragm.
b.s. = blood sacs in liver.
L.s. = liver substance.

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under normal conditions, such a necessity does not arise. In healthy individuals, the diaphragm is strong enough, even with the individual in the dorsal decubitus, to maintain a sufficient negative pressure in the chest. In this position, the pressure between the liver and the diaphragm must be positive, not negative. For, owing to the concavity of the spine in the dorsal region, the liver, in virtue of its weight, tends to sink into the chest; and the effect of the tone of the transversales themselves still further exaggerates this tendency.

Similarly, we know that the pressure of the liver against the diaphragm in all pronograde mammals is positive. Owing to the obliquity of the pelvis and the relative high position of the pubes in these animals when standing, and to the vertical depth of the chest, the recti abdominis (in this position) pass forwards and downwards to the sternum and ribs. Thus, the floor of the abdomen inclines downwards to the chest; and the abdominal viscera naturally tend to fall away from the pelvis towards and into the chest. Thus, in virtue of their weight, and of that of the viscera behind, the epigastric viscera press against the diaphragm—which in these animals is more obliquely placed than in man, passing much more downwards and forwards to the sternal extremity. But the liver and other epigastric viscera not only press against the diaphragm in virtue of their weight and of that behind, but are pressed against it much more by the tonic contraction of the abdominal wall. The recession of the recti, so conspicuous in many of these animals when in good condition and not pregnant, as for instance in dogs, due to the tonic contraction of the transversales which brace the viscera upwards and press them into the thoracic base, is sufficient evidence of this.

This (positive) pressure is of service to these animals: it not only facilitates the flow of blood through the viscera

themselves (influencing the capillary circulation), but materially assists the passage of blood from the abdomen into the thorax. How absurd is it to suppose that in a more advanced type of animal, in which the erect posture is the one of greatest activity, this pressure, instead of being greater (i.e. more positive) as we should expect, is actually less positive or even negative. We have every reason to believe that in the healthy man standing erect, the pressure between the liver and diaphragm is not only positive, but is greater (more positive) than when he is lying down: that though when standing erect, the liver is lower in the body, this position is due, not just to the effect of gravity upon the liver and other viscera, but much more to the effect of the tonic contraction of the diaphragm, which in this position is greater. The abdominal wall (though more bulged) is also more contracted. Situated between these two forces—the one above, the other below—which now are greater—the abdominal viscera are more compressed: thus, more blood per unit of time reaches the heart; and more passes through the brain and through the muscles of the trunk and limbs—an important provision, for on the enhanced activity of these organs the security of the individual in this position absolutely depends.

2. The Displacement of Organs, generally considered

It is these forces which preserve the positions of the viscera, just as a change in one or other of them causes a change in the positions of related and of more distant organs and consequently a change in the shape of the abdomen. This change in shape results from physical necessity—because the abdomino-pelvic cavity is filled with viscera, visceral parts and contents, and visceral connective tissue; because this visceral mass possesses volume, and though deformable is (except for the gas within

the intestinal tract) incompressible. Thus, even though much more should be included in the vascular pedicles of organs than the mere vessels and their fibrous coats and sheaths; even though we admit the existence of those parietal condensations of the pelvic visceral connective tissue which are known as the so-called visceral ligaments, and concede that they and other parts of this connective tissue contain much smooth muscle; yet are we unable to admit that these structures suspend the viscera or that the visceral positions depend upon them.

We cannot believe that prolapse of the liver is caused by a failure of a suspending device; but to a failure of the forces acting from below which normally maintain the liver at its high level in the abdomen. Gastroptosis is not due to a primary yielding of the connective tissue attachments of the stomach; shortening these attachments for the cure of the condition, certainly by itself, is a worthless procedure. Gastroptosis is the result of a persistent atonia of the belly-wall muscles, which allows the organ especially when loaded to sag downwards—gravity coming into play when the normal forces are reduced. The stretching of the lesser omentum is a secondary effect. The same applies to the kidneys and to other organs. We cannot believe that prolapse of the kidney is due to a relaxation of the sheath of the renal artery or to a change in the renal vessels or whatsoever may be contained within or comprise the pedicle of the kidney; but to the repetition of forces in the past, acting from above, which have gradually forced the kidney downwards with a contemporaneous lengthening of its attachments.

The mechanism of this displacement is of interest; and, being in line with that of other displacements, it emphasises our contention, supporting it from analogy. The lifting of weights is an important ætiological factor; but other

strains doubtless cause prolapse of the kidney. Lifting from the stooping position seems especially to be associated with its causation. In this position—as assumed by nurses when about to lift a patient, in which case both fore-limbs are to be used in the movement to come—the tone of the belly-wall is reduced, and consequently the intra-abdominal pressure is diminished. But the movement of lifting causes a great contraction of the diaphragm, which is rapidly induced. A great rise of pressure occurs, appearing in the sub-diaphragmatic region, which is propagated to other parts of the abdomen (and pelvis), where it is met by the resistance of those parts.

It is the great increase in contraction of the diaphragm (when the belly-wall is at a disadvantage, as it is in the stooping position) which displaces, or tends to displace, the epigastric viscera during the lifting. But the direction of the displacement and its extent, and the implication in this movement of the kidneys, depend on other factors — on whether the chest-base is free to expand and on whether the lower abdomen is well supported. If the thoracic base is free to expand (as in man), and the lower abdominal wall is in good condition, the epigastric viscera are displaced more forwards than downwards, and the downward movement of the kidneys is inconsiderable and within physiological limits. But if the thoracic base is constricted by corsets or by bands (as in women), and especially if at the same time the lower abdomen can bulge, as is often the case when ill-fitting or worn-out corsets are used, or if it bulges even slightly —which very few corsets can prevent—this mechanism is interfered with.

In this case, the displacement of the epigastric viscera is much more downwards or is altogether downwards; and the kidneys are much more affected. They are displaced downwards to a much greater extent than normal;

because, first, the epigastric viscera above directly impinge on them and thrust them bodily downwards—an impaction which does not occur when the epigastric viscera can advance. But especially is this so because the posterior parts of the diaphragm and (in this case) the related sub-diaphragmatic viscera descend much more than the parts in front—the result of the much greater length and mass, and therefore strength, of the posterior parts of the diaphragm (crural and arcuate segments) as compared with the muscular parts of the diaphragm in front. When the epigastrium can bulge and the costal arch widen, the sub-diaphragmatic viscera rotate much more: whilst the arcuate and crural fibres pull them downwards, the costal fibres pull them forwards (see Fig. 12, p. 54); and their lower surfaces, related to the upper poles of the kidneys, glide forwards over these parts, the kidneys escaping from the full effect of the displacement of the viscera above them. But when the thoracic base is constricted, the rotation of the epigastric viscera is less, the gliding movement of their lower surfaces forwards over the upper poles of the kidneys is prevented: instead of gliding over the kidneys, the posterior parts of the epigastric viscera press directly downwards upon them, and thus the kidneys are forced to descend to a corresponding extent.

This movement occurs in spite of the connective tissue attachments of the kidneys, of the tone of the tissues comprising their pedicles, and (of more importance) of the tone of the fat-laden connective tissue cells forming the beds in which the organs lie. The force of the contraction of the diaphragm, in the case considered, is incomparably greater than the force resisting the displacement which these tissues collectively offer. But it is clear that, in spite of the contraction of the diaphragm, the movement can only occur provided a space below, amidst the lower-lying viscera and connective tissue, is obtainable into which the kidneys

can be pressed: for upper organs cannot be displaced downwards unless they can be insinuated into a place below—into a space, that is, of a capacity approximately equal to their volume. The more easily this place can be obtained, the more easily will the displacement occur. Thus, the lesser the volume of the visceral mass below, i.e. of the viscera and connective tissue, etc.—the thinner the patient—the greater the liability to the displacement. And clinical experience, as we know, bears out the existence of such a relationship.

But this condition of thinness does not stand alone; for the extent of the displacement at any one time depends on the state of the lower abdominal wall and the manner of its protection at that time. As a rule, the thinner the patient, the less developed is the lower abdominal wall: but in robust, corpulent people, the muscles of this wall are hypertrophied. Thus, the effect of lifting a weight on the position of the kidneys, when constricting corsets are worn, is necessarily greater in (healthy) thin women than in (healthy) fat: for not only is the lower abdominal wall in the latter much stronger of itself than in the former, but the corsets themselves are able to support this part much more efficiently in the fat (in whom the lower abdomen bulges) than in the thin (in whom the lower abdomen is flat or even carinated). Clearly, if a lifting movement is made with the thorax constricted at the waist, and the lower abdominal wall is so supported that it cannot yield, a displacement not only of the kidneys but of the epigastric viscera themselves is prevented or is inconsiderable: for the support below prevents any bulging, and thus prevents a space being made amidst the lower viscera by the kidneys or indeed by other organs above (e.g. stomach), however much they may tend to be depressed.

But in connection with the more common occurrence

of prolapse of the kidney in thin than in plump people, there is another point to notice. The thinner the patient, the less developed the abdominal wall, the lower is the average intra-abdominal pressure; and in consequence of the lowered metabolism associated with this (a relationship to which we have referred), the tone of all the tissues of the visceral mass, especially of the fat-laden connective tissue cells, is lessened, and displacements naturally occur more easily. But the fatter the patient, the better developed the musculature of the abdominal wall, the higher is the average pressure; and with the enhanced metabolism dependent on this, the greater is the tone of all the cells comprising the visceral mass, especially of the fat-laden connective tissue cells which form beds for the kidneys, and consequently the less easy is any displacement. In the case before us, the infra-renal parts of the visceral mass resist with greater force their own deformation or displacement, and consequently the displacement of organs above them downwards amidst them is less easily produced.

These several conditions, considered collectively, explain the mechanism of prolapse of the kidney and the peculiarities of its incidence—its more common occurrence in women than in men, and in my experience in single than in married women of the third decade and onwards. There remains to mention the cause of the more frequent occurrence of prolapse of the right than of the left kidney. Whilst this may in part be due to the more frequent use of the right arm in movements of violence—lifting and over-reaching (stretching)—there is another and better explanation. Since the liver is less readily deformable than is the stomach, it is easy to understand—the mechanism causing descent rather than advance by rotation of the epigastric viscera being in play—that whilst descent of the liver posteriorly can hardly avoid displacing the right kidney downwards, causing a greater tension and stretching of the connective

tissue attachments of this organ, forced descent of the stomach is much more likely to result in its own marked deformation with a consequent less effect on the kidney of its side (Keith).

Thus, we see that prolapse of the kidney is due to the repetition of forces acting from above, when the organ is insufficiently supported by the parts about and beneath it. It is not due to a relaxation of the connective tissue attachments of the kidney (peri-renal fat and fascia). Indeed, the thickenings which occur in this connective tissue, in association with the prolapsed organ, are more or less peculiar to cases of displacement. They are not found when the organ is not pathologically displaceable. In " prolapse " of the kidney, they are the result—obviously —of the (often wide) movements which occur ; * and thus come into line with the thickenings of the visceral connective tissue found in the pelvis (p. 101).

Nor when we come to the other displacements, do we find a different mechanism at work. Hernia is not caused by a relaxation of the mesentery which contains so many vessels : but by the frequent application of a force which thrusts violently against some potential aperture or weak spot in the internal superficies, some viscus (e.g. intestine, bladder or ovary) or some part of the visceral connective tissue (as of the sub-peritoneal connective tissue or of the omentum) which may already be lying against it. It is the giving way of some such weak or inefficient place in the limiting wall, the result of repeated visceral impactions, which allows the escape of visceral parts abutting upon it, through it, with the production of hernia.

* " . . an unduly movable kidney contracts adhesions to the perinephric fascia which check, but do not prevent, movement " (Billington[8a]).

In the same way, " the erroneously called " " Suspensory ligament of the kidney " becomes developed.

The common sites of such are the femoral and inguinal regions; and that hernia should occur here is easily explained. For impactions from above not only tend to drive—as we have seen—viscera downwards into the pelvis, but act also on those more laterally placed. These are thrust downwards in the cleft formed by the abdominal wall in front and the ilio-psoas muscles behind, and impinge upon the femoral and complicated inguinal regions. If the incompressible fat in the femoral canal has disappeared, escape through the femoral ring may occur; if the lower sphincteric fibres of the internal oblique and transversalis are poorly developed an inguinal hernia may develop. The peritoneum is bulged into and through the respective apertures by the impactions of the gut or other part: the impactions cause the peritoneum to stretch and bulge, and with its bulging to displace the tissue layers external to it. In the same way is the mesentery or the other connective tissue attachments of a herniated viscus stretched: the stretching of this is caused by the visceral impactions which displace the viscus; it clearly is a result, not a cause of the displacement.

But when, in the presence of such impactions, the pelvic floor is weak or inefficient, prolapse of the uterus or of some other pelvic viscus (e.g. bladder) is much more likely to occur than hernia of other parts in other regions. The escape of some part of the visceral mass through some part of the limiting wall containing that mass, relieves the pressure and diminishes the likelihood of escape elsewhere: but multiple herniæ occasionally occur, as does hernia with prolapse. In this case, too, as we have seen, we are unable to believe that descent or escape of a pelvic viscus is due to a primary relaxation of the perivascular sheaths or of the other connective tissue attachments of the organ displaced: but we believe that prolapse is caused in a way

similar to the other displacements referred to, and that the elongation of the pedicles of the prolapsed organ is a consequential and secondary change.

3. The Insufficiency of the Visceral Connective Tissue Hypothesis: and the Essential Evidence on which the Opposing Idea is Based

Such a conception of the Statics and Dynamics of the Viscera is based on the anatomical fact that the abdomino-pelvic cavity is filled with viscera, visceral parts and connective tissue, etc., all of which either abut against each other or against the limiting wall of the cavity containing them: and on the further fact that manometric observations show that a pressure relationship exists between individual organs and between them collectively and the walls of the container. But when we come to consider the other hypothesis—that the viscera are all suspended by bands—we find nothing to recommend it. It is true that during the "evolution" of the displacement, the connective tissue attachments present an obstacle (but not the only obstacle) to its complete and immediate attainment: but this is only because the normal physiological limit of the movement has been exceeded. The displacement is a result, not of a primary yielding of bands, but of a yielding, either of visceral parts about the organ being displaced (as in the case of the kidney), or of parts of the limiting wall (as in that of hernia and prolapse), which under normal conditions should not have yielded. Even though this is so, we know quite well that these connective tissue attachments or bands cannot prevent the progress of the displacement; for with the repetition of the causes producing it, a consequent and corresponding change in the disposition and lengths of these bands occurs.

It is indeed only when one comes to realise the existence of the intra-abdominal pressure, its persistence and variations, its magnitude and frequent great rises, that one begins to perceive how hopeless is the conception that the thickenings of the visceral connective tissue maintain the viscera in position. Clearly, if the connective tissue thickenings, perivascular or other, support the uterus and other pelvic viscera, they must necessarily oppose this pressure—for they are exposed to the same vicissitudes in this respect to which the viscera are subjected. We have however but to imagine, in the living body, the soft parts below the uterus and its fascial network removed, so that the bony outlet of the pelvis—which measures four inches by five—instead of being occluded by the pelvic floor, presents as a large hiatus, and there is nothing between the organs and their connective tissue attachments and the outside air; and to think of the play of the pressure variations occurring within the abdomen and propagated to the pelvis during active life, to perceive how untenable is this idea.

Is it possible to believe that under such conditions, the visceral connective tissue, in virtue of its thickenings, could oppose the intra-abdominal pressure? Could it withstand the great and sudden rises due to cough, the great and sustained pressure produced by straining, and the rapidly varying excursions occurring with general activity? Could it sustain even the pressure which is constantly bearing upon the pelvic viscera, during posture and rest, to which all these others are superadded, and are constantly recurring? The occurrence of congenital prolapse in cases of spina bifida points to the answer. The paralysis of the levator ani muscles in this condition, shows that these muscles in the normal, in spite of their state of development in the new-born child, can prevent and do prevent, extrusion of the pelvic viscera

when the intra-abdominal pressure is raised by crying, which act causes the prolapse in these cases.

Obviously, this congenital prolapse is not to be explained by the delicate condition of the visceral connective tissue in the pelvis of the infant—it is the paralysis of the pelvic floor muscles which explains it. Can, then, the visceral connective tissue in the adult, because parts of it, at least, are denser, resist the much greater intra-abdominal pressure of that state? If this be so, injury to it, just as its supposed disease, should result in considerable changes in the positions of the pelvic viscera—visceral descent should occur and visceral extrusion be expected.

Experiment, however, does not support this idea. Removal of this connective tissue and its thickenings is not followed by prolapse. Thus in cases of carcinoma, if the uterus be removed by Wertheim's method, in which not only the uterus but the upper part of the vagina, and as much as possible of the visceral connective tissue in the pelvis, including its thickenings, is also removed, the operation is not followed by prolapse. There is no descent, no prolapse of the bladder or rectum; nor is the part of the vagina left, invaginated by descent of the intestines above. And, clearly, removal of the uterus does not affect the pressure conditions in the abdomen, nor the degree nor the manner of functioning of the visceral thrust. With the return to active life which many of these patients enjoy, the magnitude and the frequency of the considerable rises of this pressure are the same, and affect the remaining pelvic viscera as before.

How, then, is this pressure and its variations borne? Are we to believe the normal thickenings are replaced by scar tissue, which continues this service? I do not think so. Scar tissue yields to continuous tension: although it yields less readily than normal connective tissue, it

yields none the less provided the tension be considerable enough, and of sufficiently prolonged duration. It is true the mobility of the uterus is diminished as a result of inflammatory disease in the pelvis and of the scar tissue formed. Such diminished mobility is seen in cases in which scar tissue is present in the base of one or other broad ligament, such as is often found on the left side associated with the healing of a deep cervical tear produced at labour.

But this does not show that the scar tissue supports the uterus, or that it can oppose the intra-abdominal pressure. It simply shows that the normal connective tissue on the other side gives way more readily than the scar to variations of pressure whereby an adaptation to the new position of the uterus results. All that scar tissue does is to anchor the poised uterus more or less firmly to the pelvic wall to one or other side. It cannot prevent its prolapse: prolapse of the uterus occurs in patients who have suffered from such tears even when they have healed by granulation, if the causes of prolapse subsequently arise. Even a more extensive inflammation, from which many adhesions between the pelvic viscera have resulted, does not suffice to prevent a subsequent extrusion [34]. Indeed, we see too often the yielding of scar tissue, for instance, in the recurrence of prolapse after operations on the connective tissue thickenings themselves for this disease. And we see it in the yielding of the abdominal wall in the regions of surgical wounds: and especially when these have healed by granulation, that is, when suppuration has occurred, by which much scar tissue is formed, is the subsequent tendency to incisional hernia great. To prevent these herniæ, obtain healing by first intention—in other words, with as little scar tissue as possible!

Such explanations are vain. The reason the pelvic

viscera are retained in the pelvis after mutilation of the visceral connective tissue, is that there is another mechanism at work—at first sight, no doubt, more subtle—perhaps, at first, obscure; but becoming on examination as plain as the sun on a cloudless day at noon, and as simple in its action as can be. It is an exceedingly effective mechanism: for it has been produced in the course of evolution—having its origin right away back in the fishes, and becoming gradually more and more developed as it passed through the various succeeding Vertebrate Orders, as the necessity for it became more and more insistent, to find its highest development and adjustment in man. With such a history, which shows how stable its structure and how important its function must be, it is not surprising this mechanism should be able to perform the work for which it was called into being and has continued to evolve, and to perform this work efficiently—the work, namely, of successfully resisting the constantly changing pressure in the pelvis.

And this mechanism, naturally, is still present after hysterectomy for carcinoma; nor has the operation affected it one whit. On the other hand, we know too well that when the uterus, in cases of its prolapse, is removed, subsequent extrusion, not of course of the uterus, but of parts of the remaining viscera, too often occurs—the disease persists. Why is this? Is no scar tissue formed in this case? Or is the scar tissue different in nature from that formed in the other case—or less in amount or in extent? Or why? Clearly, the reason the prolapse returns is that the pelvic floor is still inefficient—it is in the same state as it was before the uterus was removed—the operation has not affected it—its condition explains now, as it did then, the existence of the disease.

The great rises of pressure caused by cough, as also the

increase produced by other activities, as well as the continuance of the pressure during posture and rest, are due, as we have said, to the increased or tonic contractions respectively, of the abdominal wall and thoracic diaphragm which compress the viscera. These viscera, as we have said, completely fill the abdomen. They extend from the summit of the thoracic diaphragm throughout the abdominal cavity, pass into the pelvis and abut upon the superior surface of the uterus and the posterior wall of the pelvis; and it is through them that the pressure produced in the abdomen is propagated to the pelvis and thus reaches the uterus. It is obvious in the intact person, that the pelvic floor musculature, which forms an obturator for the pelvic outlet, and which is situated below the uterus and its "retinaculum"—separated from these only by incompressible tissues—by contracting simultaneously with the abdominal wall and thoracic diaphragm, as palpation in the living easily demonstrates to occur, affords a much more efficient mechanism for successfully withstanding this downthrust than the radiating cervical connective tissue strands, or the perivascular sheaths of the visceral vessels which only come into play as their tension is increased by the visceral descent, and even then are only capable of passive resistance (see Fig. 22, p. 263).

We have but to consider the structure, the manner of working, and the functional capacity of the tissues of these two different mechanisms, to be sure of this. The cells of the one, we find, are eminently adapted for opposing the visceral pressure: the cells of the other are not adapted for it at all and have quite a different purpose. The most highly specialised type of cell in the one is the striated muscle cell; in the other, the non-striated muscle cell: and the argument narrows for the most part to a comparison of these. Striated muscle cells, of which the pelvic floor is so largely composed, are of extraordinary

size—about an inch long—with a complicated internal structure and many nuclei. They are multinucleated giant cells, each in the form of a fibre. In the pelvic floor, they are arranged for the most part in layers forming sheets, or plates, let into the pelvic outlet, which collectively and (with the visceral canals) effectually occlude it.

These cells comprise the essential element in the pelvic floor; but the muscles they form, like muscles elsewhere, possess connective tissue envelopes, more or less dense according to the development of the muscle enclosed and the pressure of adjacent parts (e.g. of the visceral mass above). The muscles themselves arise from and are inserted into the bony walls of the pelvis (which clearly are of importance): they do so either directly, or by aponeuroses —the connective tissue envelopes blending respectively either with the periosteum, or with the aponeuroses in which the muscle fibres themselves end. The state of development of these aponeuroses, as of the bony parts themselves from which the muscles directly arise, as of their connective tissue envelopes, depends on the functional state of these cells. A study of muscles, bones, tendons, aponeuroses and fascial envelopes in other parts teaches us this; but, strange as it may seem, the reverse, at least in part, has been maintained.

We know these cells are remarkable for their metabolic activity: in the living, the continuous activity of this tissue is largely responsible for the maintenance of the body heat. In virtue of their metabolic power, which is an index of their vitality and of the work they do and can do, they exert a continuous pull (not, however, always the same in degree) on the parts to which they are attached; and are capable at times of increasing that pull considerably. Of equal and great importance is their capacity for reacting in this way rapidly, by which

means alone are they able to meet rapidly occurring necessities as these arise. Besides on their structure, this activity is dependent on the activity of the central nervous system (cerebro-spinal) with which all striated muscles are directly connected. This assures not only rapidity of response, but degree of response: it assures the important condition of co-ordination in the reaction of individual muscles and muscle groups with other muscles and muscle groups—a distinguishing feature of this nervous system being that it governs in a concerted way muscles of different segments of the body so that all can react together efficiently for different purposive ends. In this way not only do the antagonistic muscle groups in the limbs act together; but in the abdomen, the activities of the musculatures enclosing the visceral mass are all related.

Thus, we have not only evidence of capability of the pelvic floor musculature to contract, but the possibility for it to do so, just so much or just so little as is required to meet the contractions of the abdominal wall and thoracic diaphragm. The persistence of this relationship during resting or static states is important. It is important to recognise that like muscle elsewhere, as long as its nerve supply is intact and its blood supply assured, the pelvic floor musculature is capable of displaying a continuous tone. As long as the blood in the arteries supplying it is of a sufficiently high pressure that the blood will flow sufficiently rapidly and in sufficient volume through the capillaries permeating its substance (in spite of its contraction), a continued contraction, even of considerable magnitude, can and does persist.

But when we come to the visceral connective tissue and its thickenings, we find an altogether different state of things. Striated muscle cells are foreign to this tissue, and we find no cells comparable with them in metabolic activity

amidst it. It consists of connective tissue cells and their processes enclosing cell spaces; and of non-striated muscle cells in strands. The former are either fat-laden, or form a "filmy-looking" material, with inconspicuous and delicate processes which connect the cells together, without however preventing their displacement on one another. Or, the cells have given rise to fibres more conspicuous, and which, aggregated together in the so-called condensations, form more or less definite masses.

Though to some extent elastic, these clearly are not contractile. They cannot shorten "at will," nor by nervous changes. Thus, they cannot brace up the viscera resting on the network they form to meet increases of pressure from above; nor can the vascular sheaths do so. These fibrous condensations can only respond to such rises of pressure in virtue of their elasticity and tensile strength. But these only come into play, as factors to be considered, with the elongation of the condensations; and moreover, these physical features are neither marked nor permanent. If, during resting states, the visceral condensations display a tone due to their elasticity, this is inconsiderable: it cannot prevent changes in the position of the related viscera due even to slight causes (e.g. quiet respiration): and with marked increase of tension the limit of their elasticity is soon reached. The fibrous condensations are nothing like so extensile as is striated muscle. Think of the pubo-rectalis and parturition! Compare the remarkably great extension of the muscle during this act with the small descent of the cervix in the non-pregnant, which forced traction with forceps can cause. But neither is their resistance or tensile strength persistent. The frequent repetition of the forces tending to cause some visceral displacement lead with that displacement to their elongation.

Nor does the presence of non-striated muscle cells

in the visceral connective tissue render this tissue more adapted for the visceral support. These, as we have seen, emanate from the viscera, and also fray out in the periphery; and are more or less associated with the visceral condensations of the connective tissue. Exceedingly minute, of a simple structure, with but a single central rod-shaped nucleus, and relatively few in number, these non-striped muscle cells form an inconspicuous element in the visceral connective tissue. In opposition to Moritz, it can be truthfully stated that they are not a prominent component, and certainly it cannot be maintained they are essential to this tissue. Even though, in the course of phylogenetic evolution, a gradually increasing content of the non-striated muscle in the pelvic visceral connective tissue has occurred, as Moritz states—a statement I have no reason for disbelieving and no wish to refute—there are yet not enough of these cells in this tissue of man to lend support to the hypothesis that by it the pelvic viscera are maintained in position. The notion that bands of such muscle exist amidst this connective tissue, extending from viscera to the parietal pelvic fascia (or vice versa), of any value in the visceral support, as Moritz would have us believe [67], is an exaggeration. They are nothing like so numerous as in the walls of the hollow organs, in which the bundles they form are collected into sheets, conspicuous to the naked eye and of considerable thickness. In the visceral connective tissue, the non-striped muscle cells are only revealed as masses or indeed at all by microscopic examination.

In this respect, then, they are not comparable with the striated muscle cells in the pelvic floor, which can be seen on dissection and felt on palpation. And whilst paralysis of the latter (as in spina bifida) shows that these are essential for the visceral retention and support,

the same phenomenon displays the inadequacy of the cells (non-striated and other) in the visceral connective tissue for this purpose. Thus, even though it be true that the non-striated muscle in the visceral connective tissue is more conspicuously developed in orthograde than in pronograde forms, this does not show that this tissue is concerned—even in a secondary way, much less primarily—with the visceral support. Such a difference, if it exist, can be otherwise explained: the greater intra-abdominal pressure in the former, associated as it is with an increased visceral (and so general) metabolism, suffices to explain it. But Moritz does not believe these non-striated muscle cells alone suspend the viscera or retain them in position: he definitely states the fibrous condensations largely participate.

That the visceral connective tissue is inadequate for the visceral suspension is not surprising when we consider the inconspicuous and negligible metabolic activity of its component parts. We could hardly expect that the connective tissue cells themselves should participate in this, since their metabolic power is so poor: it is independent of nervous influence, and dependent only on a plentiful supply of rich and well-purified blood. As a corollary to this, it should not surprise us to find that the fibres derived from these cells, and which form the major part (practically all) of the condensations, should be incapable of sustaining the frequently recurring strains which in the course of the pathological displacements would arise if they did not yield. Connective tissue cells only play a part in the maintenance of the visceral positions in virtue of their volume and incompressibility, and their resistance to their own deformation, as when laden with fat: they do so by transmitting a pressure to, not a traction on, surrounding cells. But they can only participate in this

way when their own position is assured, i.e. by the active resistance to the visceral pressure of the walls and floor of the pelvis. It is this also on which the integrity of the condensations of the visceral connective tissue depends.

The non-striated muscle cells exhibit a metabolism of a higher order; but, in virtue of their minute size and the simplicity of their structure, an index of the primitiveness of their type, it is not surprising this activity should be small. Unlike connective tissue cells, they possess a nerve supply; but this is from the primitive autonomic visceral nervous system, and only through this are they connected with the spinal cord. And their mode of action is quite different from that of striated muscle cells, as the difference in nervous connection suggests. The activity of non-striated muscle cells seems to be called into play much more by local than central causes, and to be more or less automatic and rhythmic in type. During resting or static states, they presumably possess a tone; but this tonic contraction, as in the case of the same tissue in the walls of the hollow viscera, is so inconsiderable as to be negligible: often, most probably, it is absent; or, if present, depends more on blood supply than on nervous impulses, and in any case the pull it exerts is small and easily overcome. The stretching of the viscera themselves in prolapse is evidence of this. It shows that the highly muscular walls of the organs are incapable of withstanding unsupported the pressures which occur in the pelvis.

Equally significant is the fact that the contraction of non-striated muscle is conspicuous more for the duration of its institution than for the briefness of its rise. It cannot react rapidly. If it were possible for the non-striated muscle content of the visceral connective tissue to respond in degree of contraction so as to successfully oppose the intra-abdominal pressure (as is conceivable if this tissue were present in mass), it would be impossible for it

to react in time with the variations of this pressure which the rapidly changing activities of the abdominal wall and thoracic diaphragm cause. The non-striated muscle of the visceral connective tissue doubtless is actively contractile, but the statement that it is "actively contractile" in this respect (Moritz) is so misleading as to be untrue.

Nor when we consider the origin and function of the visceral connective tissue do we obtain any other than corroborative evidence of the opinion expressed. The visceral connective tissue of the pelvis is but a passive tissue: a packing for the viscera, a bed for the vessels, a store-house for fat. It is the original tissue in which the viscera developed—that is why it contains non-striated muscle. And it readily adapts itself, in spite of its thickenings, to changes in the positions of the viscera. For it is easily deformable, except when the intra-abdominal pressure is high—its thickenings even giving way in response to pressure changes. The viscera themselves are similarly deformable, reacting to the same forces arising or existing in the pelvis. If they were firm and hard like buckets, the mechanism of their retention might be different than it is. But they are soft and yielding, easily stretching, so that their walls can pouch readily in any direction of diminished pressure, even though other parts of their structure and their connections should remain unaffected.

But manometric observations of the pressure in the rectum or bladder show that the pelvic viscera are not suspended. The conception that the uterus and other pelvic viscera, collectively, do not rest on any resisting superficies below, which such an hypothesis implies, is seen to be untenable immediately we begin to make such measurements in different postures and especially during active movements. The pressures found show—whether

the visceral musculatures participate in their production or not—that the pelvic viscera are pressed downwards on to a resisting superficies.

Clearly, if the viscera were suspended by bands, increase in weight of the parts above as by pregnancy or corpulency, or increase in compression as by cough, would cause but an increase in the tension of these bands—in the several segments of the parametric swing-bridge—rather than determine changes of pressure within the pelvic organs themselves. However much the viscera above are pressed downwards upon the bladder or rectum or vagina, the pressure within these parts would not rise as it does, unless these viscera themselves were pressed downwards upon some resisting "floor" beneath them on which they abut and rest, and which, acting with the force above, causes them to be compressed. Cough would not cause the pressure in the rectum to rise, unless the impact produced by cough and imparted to the rectum were not resisted by something beyond the rectum, whereby the rectum becomes greatly and rapidly compressed. If the impact produced by cough only caused the perivascular thickenings about the hæmorrhoidal vessels to become more tense, and if these structures stayed the displacement of the gut, no such rise as occurs would occur. Nor would greater pressures be found in the erect posture than in the dorsal decubitus, unless the rectum in the former were pressed downwards on to some resisting superficies below with a force greater than occurs in the other position.

Changes in the tension of the parametric swing-bridge, or an increase in the lateral pressure of these organs on each other alone, would not cause such differences. How, then, can organs be suspended by bands, or their mobility be limited by their connective tissue attachments? The fact that the pressure within the pelvic viscera is easily affected by any bodily change, even quiet respiration causing

variations—that the pressure mechanism is most sensitive —shows at once how erroneous is this conception. So also we see how untenable is the idea that the other abdominal viscera are similarly suspended. The viscera in the abdomino-pelvic cavity not only rest on each other "as on cushions," as Kelling said [55], but they are all compressed against each other by the tonic contraction of the muscular sheets which form the walls and boundaries of the cavity containing them.

CHAPTER XIV

THE SUFFICIENCY OF THE PELVIC FLOOR

1. The Fibrous Continuations or Connections of the Pelvic Floor Musculature and the Thickenings of the Visceral Connective Tissue

For reasons such as these, we believe the pelvic floor is essential for the visceral retention; and in the high metabolic power of its striated giant and multinucleated cells, we find the explanation of its capacity for this work. Like the cells of other tissues, these are subject to changes. During the life-history of the individual, the pelvic floor reacts, with the pelvic walls themselves, to changes of the visceral pressure. We have already seen that if the walls and floor of the pelvis are not capable of resisting the progressive rise of pressure produced by progressive increase in volume of the visceral mass—as occurs in the new-born child with the descent of the diaphragm and ingestion of food; in pregnancy when constricting corsets are worn; and with the growth of impacted tumours in the pelvis; or, if they are not capable of withstanding the frequent recurrence of great rises of pressure—caused by general activity, straining and cough; an alteration in the positions of the pelvic viscera and of the limiting walls and floor of the pelvis simultaneously occurs, with the sequence of either a physiological or pathological equilibrium. The bulging of the pelvis is either a physiological event, associated with an increase in strength of

the pelvis, whereby a sufficiency for the needs of the individual is attained, as in the course of natural growth and in pregnancy under natural conditions; or the bulging is pathological, showing itself earliest and most in the soft tissues of the pelvic floor as a descent with a dilatation of its aperture, but also ultimately causing changes in the bony walls of the pelvis themselves, and is associated with inefficiency.

Both these types of change in the pelvis, whereby its enlargement has occurred, is, as we have seen, associated with a change in the positions of the viscera—for the enlargement of the pelvis is due to the enlargement of the visceral mass and the consequent increase of the visceral thrust; or it is due to repetitions of the visceral impactions. This visceral displacement has occurred *pari passu* with the change in the pelvis; and, since the enlargement of the latter is permanent, the visceral displacement must be permanent also. It is true the positions the viscera come to occupy in the physiological enlargement of the pelvis are not spoken of as displacements; they are recognised rightly as the ordinary normal positions, but a displacement has occurred none the less. In the pathological enlargement, the visceral descent or prolapse is obvious enough. And, clearly, both these displacements are associated with a change in the connective tissue attachments of the viscera: in the ontogenetic displacement, with a change in disposition and the appearance of thickenings within this connective tissue; in the pathological displacement, with an elongation and yielding of these thickenings and a thickening of all the connective tissue elements which come to form part of the prolapsed mass and of those which ultimately come to suspend this mass.

But during both the evolution and the devolution of the pelvis, changes in the fibrous continuations or connections

of the pelvic floor musculature also take place. The change during phylogeny and during prolapse is not merely a lengthening of the muscle fibres of the pelvic floor: a complicated process occurs, much more subtle than this. Thus, the question arises: If the thickenings of the visceral connective tissue are incapable of sustaining the pelvic viscera in position because of the excursions of pressure arising in the pelvis; how is it that the fibrous continuations or the attachments of the pelvic floor musculature, or even the bones into which they are inserted, are capable of sustaining the strains which the activity of the musculature in opposing this force must inevitably cause? And this question becomes more pertinent than ever when we realise that the cause which produces stretching of the one is the cause of the stretching of the other. The condensations of the visceral connective tissue stretch when the pelvic floor becomes inefficient: the cause of the stretching of the "connections" of the pelvic floor is the same—inability on the part of the musculature to withstand the pressures occurring in the pelvis.

But it is easy to see which is the essential tissue. The musculature depends absolutely on its "connections." By itself, it is useless; for its contractions to be effective, its aponeuroses and the bones to which it is related must be capable of sustaining without yielding the tensions it causes. But it is not dependent in any sense whatever on the thickenings of the visceral connective tissue. The connective tissue hypothesists must believe that the visceral connective tissue thickenings preserve the pelvic floor from strains (by suspending the viscera); and, similarly, that it is the elongation of these thickenings (as by a primary relaxation) which, by causing prolapse, causes the pelvic floor to become inefficient. But such would rob the pelvic floor of a function in the normal; and be worse than mistaking a windmill for a giant.

And how idle is it to say that the ontogenetic development of these thickenings is a natural occurrence—as though *that* were an explanation ; and that in prolapse these thickenings have become diseased. We still lack a knowledge of the supposed morbid process in this connective tissue ; and we have, as we have seen, a much better explanation. For there is nothing mysterious in the production of prolapse : it, too, follows the same natural laws which determine the ontogenetic change—the overcoming by the visceral downthrust of the resisting mechanism below, accompanied, instead of an evolution, by a devolution of its tissues. It may be said that the connective tissue attachments in prolapse have been unable to prevent the disease because the pressures causing prolapse are great ; but this also is untenable. In the normal adult, the exciting causes of prolapse are constantly recurring ; the rises of intra-abdominal pressure are as great, as intense, or even more so, than those occurring in patients suffering from prolapse ; yet in them prolapse does not arise.

How, then, is it that whilst the visceral connective tissue attachments readily give way when the viscera are insufficiently supported, the fibrous continuations or attachments of the pelvic floor do not yield in the normal ? The answer to this question is to be found in the differences in the nature of these two types of " connective tissue." These are essential, fundamental ; and, clearly, are due to the difference in origin and structure of the types. The thickenings of the visceral connective tissue are incapable of withstanding strains of any appreciable amount, because of their origin and structure : the fibrous continuations and connections of the pelvic floor, because of *their* origin and structure, are capable of doing so continuously and considerably, though in varying degree.

The difference in origin is considerable—and significant. The visceral connective tissue thickenings, as we have seen, are derived from visceral connective tissue cells of low metabolic power. But the fibrous continuations of muscle are formed from the same sort of cells from which striated muscle cells themselves arise—they are formed either by a change of striated muscle fibres (or cells) themselves, as in regression; or from cells, some of which give rise to striated muscle cells (as during early development). That is to say, they are developed from mesoblastic cells of a markedly or relatively high metabolism.

Derived from cells of such vastly different powers, it is not surprising that the functions of these two tissues—their reactions, respectively, towards strains—should be so dissimilar. In line with this, is their difference in structure. The fibres of tendons and aponeuroses are arranged in definite order, for the most part longitudinally, parallel with each other, and are clearly defined: whilst, extending throughout their length, amidst the fibrils composing them, exist columns of well-marked stellate cells, each containing a conspicuous nucleus and being contiguous with its neighbours [88]. But the visceral connective tissue thickenings show a more or less closely interwoven structure, with wavy fibrils, irregularly disposed and matted together, passing insensibly into the surrounding looser connective tissue; and their cells are relatively few and inconspicuous, and are arranged without order.

The common origin of the cells forming the fibrous continuations of muscle, and of the muscle cells themselves, disclose their common function; and explain how it is that tendons and aponeuroses, like muscles, possess afferent (cerebro-spinal) nerves. The tendon or aponeurosis and its muscle form a tensile mechanism, highly variable in activity and capable of great and rapid changes. In it the

fibrous portion plays a passive, yet the important part of not yielding, in spite of the pull of the muscular segment; and of ever being of a tensile strength sufficient for the muscle. It is the activity of the muscle which determines this: it is because the fibrous parts are associated in their function with the activity of the muscle that tendons and aponeuroses possess such strength.

The activity of the muscle, on which its own developmental state depends, affects the developmental state of its tendon or aponeurosis—presumably by affecting the cells in these structures. With hypertrophy of muscle, a corresponding hypertrophy of its connective tissue attachments occurs; but these become thinner and weaker as the muscle weakens. This is a matter of nutrition. With an increased activity of muscle, such as leads to its hypertrophy, its blood-supply becomes greater and the nutrition of its tendon is correspondingly increased. The tendon cells being better supplied, not only continue secreting or laying down the necessary fibrils, but do so in increasing quantities and give rise to new columns of cells which lay down other fibres. But with the inactivity of muscle, such as leads to its atrophy, the blood-supply is diminished, and a corresponding fall in the nutritive supply to the tendon cells occurs, the activity of which correspondingly dwindles.

Such an effect is not without parallel in other apparently passive tissues. The skeleton is similarly influenced. In the life-history of man it plays a passive part, but can scarcely be considered a passive tissue since it contains remarkable cells. Its form is caused not only by inherent developmental tendencies, and modified by the activity of "internal glands;" but also by the play of pressures and tensions, caused on the one hand by forces of the environment, and on the other by the reactions of striated muscle. The form of the pelvis, as we have urged, is

related on the one hand to the visceral thrust and its excursions; and on the other to the pull of the muscles of the pelvic floor.

Pressures and tensions affect its parts by affecting the nutrition of these parts. A "rarifying osteitis" results from the exposure of bone to continuous pressure above a certain amount; the rate of absorption depending on the degree of that pressure, i.e. on the possible response of the bone to it. Bone disappears rapidly in front of an enlarging aneurysm: during pregnancy the pelvis slowly enlarges. Only when the nutrition of bone is not interfered with by such pressure is an equilibrium maintained. Greater variations of pressure than normal, if often repeated, by increasing the nutrition of bone (by increasing its blood-supply), lead to its consolidation (or hypertrophy); but, in the absence of ordinary variations, bones remain or become weak. Striated muscle affects bone to which it is connected in the same way that it affects its tendon or aponeurosis by which in many cases that connection is made. By its own activity, striated muscle influences the activity of the osteoblastic cells —the wonderful corpuscles. The greater the pulls, the greater the reaction of the bone, the more marked the place of insertion and the stronger the bone becomes. But, with long lasting inactivity, the muscular marks gradually become effaced and with this ensues a local atrophy. Such changes are explained by the nutritive changes which the activity of muscle locally occasions.

The case of the condensations of the visceral connective tissue is essentially different from that of tendons and aponeuroses. They are not associated with the pull of striated muscle, but with the displacement of organs to which they are related. When this displacement is restricted by the maintenance in their positions of the

surrounding incompressible visceral parts by the efficient resistance of the walls and floor of the pelvis, a tension in these attachments can hardly, or cannot occur. Under normal circumstances they are not subject to strains, or to strains of any extent; and certainly, they are unused to reacting towards or dealing with such strains. Thus in them the very opposite condition obtains to that of tendons and aponeuroses, in which the sustaining of such strains is the normal state of affairs. It is only when the resisting surrounding parts give way that a visceral displacement of magnitude is possible; and it is only in such case that unusual strain falls on the connective tissue condensations.

But a reaction to this—even in the case of the visceral connective tissue—nevertheless occurs; but it only does so to any extent late in the disease, when the pelvic floor has become so inefficient that it is unable to maintain the prolapse in place and prevent further extrusion. Even so, it does not suffice to stay the disease, nor to permit as high pressures in the pelvis to occur as before, for this reaction is of a different order to that occurring in tendons and aponeuroses with hypertrophy of their muscle. With the stretching of the so-called visceral ligaments in prolapse—an atrophic process for which the continued stretching, inevitable with prolapse of the uterus, readily accounts; and which, in no other sense, is a disease — adaptative changes in the pelvic floor tend to take place in the endeavour to procure and maintain a state of equilibrium (see Fig. 25, p. 351). In cases of partial prolapse, and as long as this condition persists, the so-called visceral ligaments, still intra-pelvic, as we have seen, do not thicken. It is only when the pressures in the pelvis are too great for the muscle, and the prolapse in consequence is becoming complete, that a collective thickening of these ligaments

(as of other tissues) sets in. Whilst in the former case, the prolapse is suspended by the grasp of the pelvic floor musculature in the region of the aperture, and the parts within the pelvis are relieved of all strain; in the latter case, the conditions are reversed.

The parts of the visceral connective tissue not before condensed and now only beginning to be subject to strains, thicken; as do the attenuated ligaments. As in the ontogenetic displacement, so here the still cellular (intra-pelvic) connective tissue reacts to the tension now arising within it. But whereas in the former case, the pelvic floor also reacts so as to support the enlarging pelvic visceral mass more and more efficiently, and so limits the strains to which the thickening connective tissue is subjected; in the case of advancing prolapse, the reaction of the pelvic floor is either inadequate for this purpose or has become altogether incapable of it (Fig. 3, p. 18). Similarly, the connective tissue and other elements (e.g. epithelial layers) in the prolapsed mass thicken to a greater extent than before. And just as the thickening of the visceral connective tissue still within the pelvis is due to the strains put upon this tissue by the extruded visceral parts, now no longer suspended by constriction; so the latter is caused by the reactions of the tissues comprising the prolapsed mass to the variations of pressure occurring within that mass, which are greater than before. For the pelvic floor aperture is now larger, and the parts prolapsed are less preserved from pressure impulses; and being unsupported below, they have of themselves to meet the variations of pressure transmitted to them.

It is because the parts of the visceral connective tissue which thicken in prolapse, with their transformation, do not take on the form and structure of tendons or aponeuroses, a type of tissue capable of bearing strains, that the

thickening which occurs is insufficient to stay the disease, except in the latest stages. It may be said that these thickenings retard the advance of the disease, just as the thickening of the superficial parts of the prolapsed mass (e.g. of the everted vagina) tends to sustain the pressure within. But in the early stages of prolapse, the pelvic floor still functions, and the part about the aperture, by embracing the enlarged and pathological obturator, tends to check further escape; whilst in the later stages—when the disease is greatly advanced—and the thickening of the prolapsed mass itself and of its intra-pelvic connective tissue attachments is considerable, the causes which produced the disease have died away.

The manner of living in such cases has entirely changed. The intra-abdominal pressure is now nothing like so high, nor are the variations due to activity of the same extent as before. The thickening of the visceral connective tissue in prolapse may be regarded—quite fairly—as an attempt to re-establish a state of equilibrium; but the equilibrium is at a much lower level than normal. This is shown by the disabilities of patients with advanced prolapse. These women are rendered incapable not merely because they have a mass protruding between the legs, but essentially because they are not capable of bearing—or of producing—the same degree of intra-abdominal pressure as when well, which is necessary for and occurs with activity. Not only are the parts of the mass prolapsed, though thickened and "stronger," still incapable of playing the part striated muscle plays and of withstanding the pressure incidental to health; but—much more important—with the atrophy of the pelvic floor which ensues, a corresponding atrophy of the abdominal wall and of the thoracic diaphragm has certainly occurred. Look at Figure 3, and imagine the state of the abdominal wall and diaphragm

belonging to such a case! It needs no divination to say they also were degenerate.

2. The Pelvic Floor and its Lining

It is perhaps not remarkable that authors—even those who consider the condensations of the visceral connective tissue of essential importance for the visceral support—should yet credit the pelvic floor with a share, certainly a subsidiary one, in that work. Even Fothergill admits the pelvic floor has a sphincteric function or action on the visceral canals as these pass through its aperture. But he will not allow that the part of the floor behind the aperture and at its sides—the broad superficies which extends from one ischial spine to the other, and from the end of the sacrum to the lateral pelvic walls anteriorly on each side of the aperture—plays any part in retaining the pelvic viscera in the pelvis. He thinks this part is functionless—degenerate—vestigial. Thus, for him, the anatomical relationship between the saucer-like concavity of the pelvic floor and the pelvic visceral mass abutting upon it, is without physiological significance.

Martin's idea is different. It cannot be denied, he says, that the pelvic floor participates in the maintenance of the pelvic visceral positions and even to a certain extent understruts the condensations of the visceral connective tissue. "Thus, we can speak of an upper and a lower supporting apparatus—the retinaculum uteri and the pelvic floor. But from clinical observation," he adds, "one can only attribute to the latter a subordinate role" [61]. The majority of authors are in like case; for them the anatomical relationship between the pelvic floor and the pelvic viscera collectively is too close to pooh-pooh the possibility of a pressure relationship.

But very different opinions concerning the extent of

this relationship have been held. Some see in the pelvic floor but an auxiliary which comes into play during time of special stress, e.g. during the carrying of heavy burdens, etc. (Griffith, [32]); others see in the "levator ani . . . the most important factor in the retention of the pelvic . . . viscera . . ." (Stevens, [96]). The tendency is to assign to the pelvic floor a more and more important share in this work. Its paramount importance is not yet allowed. This is because the intra-abdominal pressure, its variations and significance, are scarcely appreciated; and because the reactions of striated muscle in this respect have not been considered. Certain it is that had these been sufficiently studied, the full significance of the relationship between the pelvic floor and the pelvic viscera would long ago have been recognised.

The pelvic floor is for us but the musculature, which made up of individual muscles, occludes the pelvic outlet. We hold this view because we see in the musculature the real supporting mechanism—the actual floor. But the fascial sheets lining the internal aspects of the muscles, separating the muscles from the visceral mass, may also be regarded as part of the pelvic floor. But when we think of a floor, we think of a supporting surface; and the fascial lining of the floor can only be considered as a part of the floor if it be supposed to participate in sustaining the pressure of the visceral parts pressing downward upon it. By the majority of authors, this fascial lining is so regarded. Some, perhaps most, regard it as more important than the subjacent musculature—thinking it participates chiefly or largely in the work of retaining the viscera in the pelvis; acting either with the muscle below it, or with the condensations of the visceral connective tissue above it, or indeed with both, according to the particular opinion of the individual. Thus, Martin says that though the pelvic

floor participates in the visceral support, . . . "it does so more in virtue of its fascial lining or sheath than in virtue of its muscular substance" [61]. And those who believe the levator ani is but an auxiliary to meet extraordinary pressures in the pelvis are of the same opinion. Thus, Griffith is of the opinion that the general pelvic fascia, which forms a continuous sheet, varying in density in certain positions, is the chief support against ordinary conditions of pressure [31]. Cameron, too, believes the pelvic sheath of the levator ani is of more importance than the muscles [14].

The fascial lining of the pelvic floor, which is nothing less than part of the parietal pelvic fascia, is but the intra-pelvic parts of the fascial envelopes enclosing the levator ani and coccygei muscles. It is strange that these should be considered more important than the muscles to which they are related. In the case of the limbs, the state of the development of the fascial sheaths of muscles depends, as we have mentioned, on the functional activity of the muscles concerned. Similarly, it is plain, the fascial lining of the pelvic floor is dependent on the activity of its associated musculature. This fascial sheet only arises during ontogeny because the pelvic floor—the musculature beneath it—is able to persistently adapt itself to the variations of pressure and the increasing demands on it which with growth occur. If a child with spina bifida and resulting congenital prolapse could grow up (uncured), the fascial lining of the pelvic floor would obviously be different in the adult state to what it is.

The fascia lining the abdomen (transversalis fascia) and the parietal pelvic fascia are similar in this respect. They are the result of the embryonic connective tissue matrix, lining the muscular parietes of the abdomino-pelvic cavity, gradually becoming condensed, as a result of each becoming subject to lines of stress

by being displaced and so stretched. Such of course begin to appear, or to become prominent, immediately after birth: lines of stress are produced in these strata of connective tissue whenever the belly wall bulges and the pelvic floor descends, and thus occur to some extent with every respiration. They are caused by the variations of the intra-abdominal pressure, in the production of which the musculatures to which each is related directly participate—each bulging or receding according to its own contraction and the contractions of the other muscles enclosing the visceral mass and compressing this mass in an antagonistic way from elsewhere. Since the musculatures concerned limit the displacement and consequent stretching of their connective tissue linings or internal fascial sheaths, it is plain the activity of these musculatures is of essential importance in relation to the developmental state of these linings or sheaths. Thus, if the fascial lining covering the pelvic floor appears to play a part in the mechanism of the visceral support, it is only because it lies superjacent to the musculature by which it is kept in place.

The evidence in support of the opposite view is extraordinary, or wanting. Martin takes us to the abdomen. Incisional hernia, he says, only occurs when the suture of the fascia for some reason or other has not held. "The result of operation for hernia will always be bad, if one does not succeed in uniting the fascial margins" [61]. Griffith gives no reason at all for his opinion. Cameron's argument is plainly erroneous.

Martin's references to hernia are not pertinent to this question. He forgets that the fascial layers dealt with during laparotomy and in operations for hernia, are not the fascial sheaths or envelopes of muscles—though they may serve that purpose (as in the case of the recti)—but their

aponeuroses of origin or insertion: * and he forgets that the condition of these depends on the nutritive state of the muscular sheets they continue. All the world recognises the importance of the muscular tissue in the abdominal wall—especially of the flank muscles—though the ultimate reason of this importance may not be known. Surgeons, when opening the abdomen, separate longitudinally whenever possible the fibres of a muscular sheet rather than divide them transversely. Surgeons take this care, because they know that incisional hernia is much less likely to develop in the former than in the latter case, however carefully the "fascia" be sutured. For a similar reason, they most carefully preserve nerves: division of the afferent or efferent muscular nerves renders the muscle or the part of it affected, inactive, and subsequent hernia much more likely or certain, in spite of the fascia covering it (the aponeurosis of some other muscular sheet) being intact.

And for the cure of inguinal hernia, at least in the adult, it is known—past question—that the lower muscular margins of the internal oblique and transversalis muscles, arching over the canal, must be successfully dealt with. The more hopeful prognosis in cases of inguinal hernia, as compared with that of femoral hernia, depends entirely on the fact that in operating for the former the aperture can often and easily be occluded by bringing down the muscle margin to Poupart's ligament; whilst such a procedure in femoral hernia is more difficult.

* The muscles of the abdominal wall have their own specific muscular sheaths. These are quite distinct from and much more delicate than the aponeurotic continuations of these muscles. Thus, after splitting the aponeurosis of the external oblique (as for appendicectomy) one comes upon some delicate areolar tissue and then upon a thin layer of connective tissue forming the outer part of the sheath for the internal oblique. This has to be cut through before the fibres of that muscle can be separated.

In femoral hernia, the entrance to the femoral canal can only be closed in an analogous way by uniting the lower margins of the internal oblique and transversalis to Cooper's ligament (Lotheissen's operation [85])—a much better operation than closing the ring from below by suture.

Again, we know quite well that the prognosis of operation for inguinal hernia, when efficiently done (as in the way indicated) depends largely on the physique of the patient: it depends on the degree of development of the musculature of the abdominal wall (especially of the flank muscles), and on the possibility of the patient's nutrition being sufficiently good to establish and maintain a good muscular wall in the damaged region. Surgeons refuse to operate on poorly nourished individuals, because they know that even if the wound—not only of the skin, but of the fascia—heals by first intention, the hernia is likely to recur if the muscular parts of the parietes are weak and arduous work have to be done. The fascia yields (however well it may have been sutured), if the muscular part of which it is the continuation is not well developed; it yields because it also is weak when the muscle with which it is associated is weak. If the muscular part cannot withstand the excursions of the intra-abdominal pressure which the manner of living or of earning a livelihood occasion, its aponeurotic part also is unable to do so. The condition of the two invariably go together.

A good example of this is seen in the changes which take place in the abdominal wall during pregnancy. As the abdominal wall becomes stretched by the growing uterus, not only do the flank muscles give way, but also the median aponeurosis, both becoming thin and weak [75]. An analogous condition of the pelvic floor and its adjacent parts is seen in advanced prolapse. When the pelvic floor musculature has given way, by

stretching, because it has been unable to cope with the intra-pelvic pressure and its excursions, not only is it degenerated, but its aponeurotic connections also have suffered (Fig. 3). On the other hand, when these muscles hypertrophy, so do their fascial continuations. It is absurd to compare the aponeurotic continuations of muscle with the fascial linings or envelopes of muscles. The aponeurosis of the external oblique is much more than a fascial lining or covering for the internal oblique: it is the means by which the external oblique functions, and that is why it is of importance. Similarly, it is erroneous to think that the cure of hernia depends only on the suture and successful union of the fascia: it depends on much more than the closure of the aperture—it depends on the state of the muscles of the abdominal wall; on the volume of the visceral mass; on the excursions of pressure which a return to activity will cause.

Since striated muscle is such an important constituent of the abdominal wall, it is not surprising that in the pelvic floor it should reign supreme. To think that this musculature functions only during times of stress is peculiar; for this is to separate arbitrarily the striated tissue in this region, in the matter of physiological reactions, from all striated muscle elsewhere and to put it in a category by itself. I do not know the basis of this idea. It is a conception intermediate in position between that in which the muscle is denied to function at any time, and that in which it is believed to function at all times. It is perhaps based on a conception of fatigue. It is perhaps natural to think that if the pelvic floor should function continuously, even though its activity should vary, it would readily become fatigued. It seems reasonable to suppose that during resting or static states the pelvic fascia should suffice to maintain the pressure in the pelvis and the viscera in

position, relieving the muscle below of work during these times.

But people who hold such ideas have not recognised the physiological dependency of the fascial sheets lining muscles on the activity of their related muscles. And they forget that the contraction of striated muscle is continuous, that it displays a tonic contraction which though it varies under different conditions is persistent. The characteristic of striated muscle of rapidly increasing its contraction, and thus causing movement when its antagonists permit, is no more peculiar than this feature of persistent tone. And that this should occur is no more strange than that other cells should display a similar persistent activity. Nerve cells, for instance, are continuously active—e.g. those of the medulla, on which respiration depends: so also are those of the vascular mechanism, by which a continuously raised blood pressure throughout life, even during sleep, persists. We live but one life: during that life the essential cells at least of the body are continuously active, though their activity undergoes great rhythmic variations. During some phases, so great is the diminution of this, that cessation of life may almost be simulated. Thus, mothers are not unknown to have awakened their healthy sleeping child to see if it live.

The fatigue of the pelvic floor, however, has not been raised into prominence by authors; but for other reasons, notably the supposed rudimentary nature of its muscles, their exposure during evolution to retrogressive changes, it has been disregarded. For such reasons, Cameron assigns to the fascia lining the pelvic floor, in the matter of the visceral support, a more important function than to the muscle beneath it. His remarks on this pelvic sheath, as he calls it, are interesting. He thinks this sheath, instead of being a condensation of the peri-muscular connective tissue, represents the part of the

levator ani itself which in the course of the phylogenetic change from the pronograde to the orthograde type, has undergone regression. He quotes Bland-Sutton's well-known views on the subject of muscle regression [9]—that when a muscle degenerates, either from morphological or pathological causes, the amount of contractile tissue is diminished and the parts take on the character of fibrous tissue or ligament. Since, says Cameron, the pelvic sheath of the levator ani and coccygeus is distinctly aponeurotic in character as a rule, it may therefore quite possibly represent a transformation of the contractile element into fibrous tissue [14].

This, however, is untenable. For when a muscle atrophies — that is, becomes thinner — the contractile part affected is not transformed into connective tissue; but it disappears. The muscle undergoes a partial autolysis. That, in such case, the contractile substance is not added to the fascial investment of the muscle whereby this becomes thicker, is shown at once by the differences in the fascial investments of the same muscles in different individuals. Whilst in individuals with a poorly developed musculature, these are thin and poorly developed; in well-developed people, not only are the muscles themselves well developed, but the fascial sheaths of the muscles are also conspicuous. Nor in the transition from the well-developed to the atrophic state—the result, for instance, of some wasting disease, in which the muscles obviously participate—is there any evidence (but the reverse) that the fascial envelopes of muscles thicken as the muscles themselves dwindle away.

But as a result of regression, quite a different condition obtains. The muscle has not only become thinner, but shorter. The transformation of contractile substance into fibrous tissue has not occurred laterally to the muscle length

—that is, circumferentially to a muscle belly, or superficially to a muscular sheet—but longitudinally within the muscle itself; so that a muscle fibre, instead of pulling on another muscle fibre in line with it, now pulls on a tendinous fibre. Part of the muscle has disappeared and its place has been taken by fibrous tissue which continues the pull. Thus, with a diminution in length of the muscular part, a tendon or aponeurosis appears; or if such already exists, there is an increase in length of the tendinous or aponeurotic part of the muscle. Usually, this change takes place at one or other extremity of the muscle. Sometimes, it happens that it occurs within the length of a muscle rather than at its ends—a muscle belly, for instance, becoming divided transversely into two separate muscles by the formation of an intermediary tendon which joins the muscular parts together and renders each effective.

In the case of the levator ani, the part which has regressed is the part above its present line of origin. It is represented by the layer of fascia which passes upwards from the upper margin of ilio-coccygeus to the pelvic brim, above the limit of the obturator internus; and which forms the second or the inner layer of the parietal pelvic fascia found in many cases in this region (Thompson [98]). The pelvic sheath of the levator ani has nothing to do with this change. Whether this sheath is aponeurotic in structure—whether it has the structure of such tissue and is therefore capable of bearing strains—possessing long columns of active cells—is for us of little importance; for its strength, such as it may be, must depend on the underlying striated tissue. In this respect, however, it is interesting again to notice that Cameron considers this sheath as part of Mackenrodt's ligament, which if anything rather detracts from its significance.

3. The Regression of the Pelvic Floor

When one remembers that after death muscle rapidly decays and soon becomes weak and poor stuff compared with fibrous tissue—even with that derived from visceral connective tissue cells; that in the old and decrepid—in most dissecting-room subjects—the levator ani is often in an atrophic state; and, further, when one is brought face to face with the regressive changes which the pelvic floor muscles have suffered during evolution; it is not difficult to understand the general trend of opinion that the pelvic floor muscles can be of little service in the body, and indeed are but relics of the past. To counter-balance these one-sided impressions, which have attracted so easily contemporary thought down the broad, well-trodden and easy declivity of simulation, from the depths of which no perspective can be seen, we must undertake further and other investigations. To appreciate that the levator ani really exists and functions, we must leave the dissecting-room and study the living: to understand why it functions, we must begin to think about the intra-abdominal pressure and why the viscera are compressed—and especially to consider the mechanism of the capillary circulation: to refute the fallacious suggestion that because these muscles have regressed they are therefore degenerate, we must study the nature and cause of regression. Above all, we must grasp the fact—or the possibility of the fact—that in the course of evolution—in the individual as in the race—regression may come to a standstill, and because of other advancing bodily charges (e.g. *of metabolic rate*) a state of equilibrium may again be established in which the muscle previously regressing can now function adequately or even respond by hypertrophying to further changes.

The subject of muscle physiology and regression, absorbingly interesting as it is, hardly comes within the scope

of this book. We have, however, this observation to make —that the changes which occurred in the pelvic floor muscles during evolution were not simple atrophic changes. It is generally believed that the so-called retrogressive change of these muscles was due to the loss of the tail: but this is the result of jumping to a conclusion without staying to consider the matter. Were this true, but a simple atrophy of the pelvic floor muscles would have occurred: these muscles would merely have dwindled away; and such relics as remained, instead of arising half way down the pelvic walls (i.e. from the white line) would still arise from the pelvic brim (ilio-pectineal line) as they do in pronograde mammals that possess them. The fact that these muscles have regressed shows that some cause other than mere loss of the tail has been at work. When all the evidence is considered there can be no doubt that this cause was the increased pressure in the pelvis which became greater (perhaps relatively suddenly) as locomotion with the body vertical (e.g. suspended by the arms from tree branches), instead of horizontal, gradually became habitual; and that only when equilibrium had been re-established could further advance (descent to the ground and walking erect) have occurred.

Clearly, the pressure in the pelvis is greater in orthograde than in pronograde types. Even if we consider only the effect of gravity on the viscera must this be so. Much more reason have we for believing this when we take into consideration the effect of the tonic contraction of the abdominal wall muscles and of the thoracic diaphragm, which is greater in the erect posture than when in the stooping position. And when we recognise that the gradual assumption of the erect posture meant the gradual elevation of the head end of the animal, and that (if nothing more) gravity rendered more difficult the supply of blood

to the brain, the necessity for a gradually increasing intra-abdominal pressure becomes obvious. For the intra-abdominal pressure directly affects the circulation: and a gradually increasing intra-abdominal pressure occurring during evolution (such as we have reason to believe did occur) would naturally cause (besides other changes) a progressively increasing response on the part of the vascular mechanism. A phylogenetically increasing intra-abdominal pressure would have this effect by determining (at each of its stages, within limits) a better—a quicker and larger—return of blood from the abdomen to the heart and so a better output from the heart and thus an increasing aortic pressure: causing in this way a progressive improvement in the flow of blood to all the organs and body parts. Much more obvious does this necessity become when we recognise that with this evolution, the brain itself developed; and therefore required more blood per unit of time than before.* Thus, we see that with the gradual evolution of an animal conspicuous for his orthograde posture and carriage and his cerebral activity, a gradually increasing work on the pelvic floor muscles must necessarily be posited.

Such a conception presupposes plainly enough that the pelvic floor muscles in pronograde types serve the same purpose as they serve in orthograde forms as in man; but this supposition is easily justified. The conception at least has this in its favour that it brings pronograde and orthograde forms into line with each other in this respect; whilst, on the contrary, the prevalent opinion—that the pelvic floor in the former are simply tail-moving muscles and that in man their function is changed—thrusts these

* According to the author's opinion this is putting the cart before the horse: for the brain evolved because its blood supply improved—in other words the increased blood supply was greater than the immediate needs of brain.

two types asunder in opposition to all biological laws. It is to be regretted that this latter idea has such a hold on present-day opinion: for it is wrong. The pelvic floor muscles are not tail-movers—they are not associated with movement of the tail as generally understood. These muscles have nothing to do with such movements as the wagging of the tail in dogs; with its movements in cats; with its grip in South American monkeys; with its lashing movements in cattle and horses. The ischio-coccygei * (homologous with the coccygei of man), and well marked in the guinea-pig, can have nothing to do with the wagging of the tail in the guinea-pig, for the tail does not wag—there being no tail. Yet the muscles are there! How absurd is it to think that the similar muscles in cattle which depress the tail more than abduct it, cause or assist in causing its active movements in these animals. † It is the continuation of the sacro-coccygei muscles, which extend throughout the length of the tail, which cause the various movements of the tail—movements of different segments on each other, and movements in different directions; just as it is the similar muscles about the spine which cause movements of the spine. Other muscles may indeed move the spine; but they do not exist to move the spine, and the movement of the spine is, as far as they and their purpose are concerned, a secondary affair.

So it is with the pelvic floor muscles. These exist in pronograde mammals to oppose the pressure and its variations in the pelvis; and in doing so, or to do it more

* It is stated quite recently (1916) in an authoritative work on Gynæcology, that the ischio-coccygei are tail-wagging muscles.

† Cattle and horses possess no muscles homologous with the levator ani (i.e. pubo- and ilio-coccygei); the ischio-coccygei being the only muscles of the pelvic floor in these animals. In many animals in which this is so, the ischio-coccygei are well developed: they serve the same purpose as the levator ani, but on account of their attachments less efficiently.

efficiently, may move the tail. But such movement of the tail, when it occurs, is not a primary movement of the tail; it is not a movement which by itself is of importance, as for instance is that made to express an emotion or to flick flies off the flank. It is a movement for an end—the resistance of the visceral pressure in the pelvis—in which the tail and only its proximal part—the tail root—plays a secondary and passive part—the change in the muscles which cause the movement being the essential factor, and which in pronograde mammals often occurs without movement of the tail.

Nor is there any doubt that in these animals during activity the pressure in the pelvis is greatly raised. When a dog barks, one can see (in short-haired forms) the impact bulge the perineum: when a cat springs, one cannot but believe a great increase in intra-abdominal pressure is produced. And just as it is the active predacious habits of carnivora which occasion such rises of pressure in the pelvis as to necessitate such an efficient closure for the pelvic outlet as is found in them; so it is the difference in respect of the manner of living, which differentiates herbivorous from carnivorous mammals, and which explains how it is that in the former the closure of the pelvic outlet is less efficient than in carnivorous mammals. Differences in the tail, and in its mobility, do not explain this difference. The closure is less efficient in herbivora, because in them the pressures to be opposed are never so high as in the other type of animal: the pressures are never so high, because herbivora never use their fore-limbs in the same way as do carnivora—they never crouch to spring, and never use their limbs to strike down prey. The absence of the pubo- and ilio-coccygei, which in carnivora permit great rises of pressure in the pelvis to occur with impunity, even determine differences in posture: herbivora never sit on their haunches for this reason, a position carnivora often assume.

And there is no doubt that during the evolution of the orthograde type of animal, the pelvic floor muscles were greatly exercised—because of the greater variations of pressure produced by activity with the body vertical, and the persistence of a greater pressure as long as the upright posture was maintained. We cannot doubt these muscles had difficulty in meeting this pressure and its excursions; because the body was as yet unaccustomed and consequently unadapted to this new manner of living. That these muscles drew the root of the tail forwards and upwards against the perineum and maintained it there, for this purpose, is clear. Indeed, the position of the coccyx and the limitation of its movement in orthograde forms indicate that only in this way could the pelvic floor muscles successfully oppose the visceral pressure and its variations; and that a very great, or indeed a maximal contraction of these muscles was necessary for this end.

Contrast, for a moment, the conditions which obtain in this respect in the two types of animal. And what a difference do we see! Whilst the proximal coccygeal vertebræ, forming the root of the tail in mammals of the pronograde type, have a wide range of movement in the dorso-ventral plane; in orthograde mammals, especially in man, their movement is very limited indeed. A dog can hold his tail erect; extended straight backwards; or down between his legs: but man can only hold his coccyx in one position. This simply means that the pelvic floor muscles in the dog are able to play their part of a pressure-resisting mechanism, resisting the pressure in the pelvis and the impactions which occur during activity (e.g. during barking), whatever the position of the tail; and are thus able to allow to the coccyx (and so to the free part of the tail) an extensive range of movement; but that in the tail-bearing precursors of man it was otherwise.

During the evolution of the dog—a type of carnivorous

mammals—the pelvic floor muscles were able (at least from less distant times) to occlude the pelvic outlet easily whatever the position of the tail, however it were held or moved; for the simple reason that all the forbears of the dog always maintained (as the dog himself for the most part maintains) the pronograde type of posture and locomotion. For from this characteristic, the pressures in the pelvis never became so great—certainly, during the maintenance of common postures they were never so great—as in the forbears of the orthograde competitor; and thus were at all times easily and efficiently dealt with. But during the evolution of the orthograde form, the root of the tail gradually became drawn more and more downwards and forwards; because the contraction of the pelvic floor muscles had to be great, causing the proximal free part of the tail to come into apposition with the perineal parts and its distal free part (if one still existed) to protrude forwards between the legs, whilst its proximal buried part was acutely flexed on the scarum, as for instance it is in the new-born child (see Fig. 16, p. 93).

And we have other evidence that this was so. If we take a cat and hold it vertically by the scruff of its neck, its tail naturally takes this position. If we pull the tail away and then let it go, it returns immediately against the perineum. The reason of this is that owing to the effect of gravity on the blood stream, the vertical position requires, for the same blood supply to the brain as in the horizontal position, a higher aortic blood pressure than when in this latter position; and for this the pelvic floor muscles, as those of the abdomen (abdominal wall and thoracic diaphragm), have to contract to a much greater extent. By increasing the compression of the visceral mass, they cause an increased flow of blood to the heart and an increased aortic blood pressure results. The brain is as well or better supplied with blood, and consciousness

persists. The reason of loss of consciousness which occurs on holding a tame rabbit up by the ears for a little while is due entirely to the fact that its body wall muscles are weak (and its pelvic floor is inefficient) and a sufficiently high intra-abdominal pressure cannot be maintained. Thus, we see that the acute flexion of the tail, occurring when we suspend a pronograde mammal by its neck, results from the maximal—or, at least, the unusually great—contraction of the pelvic floor muscles; which is persistent as long as the vertical position (and consciousness) continues.

Such a persistently increased tonic contraction of the pelvic floor muscles, induced by the tendency to assume orthograde habits by ancestors of man, is of itself sufficient to explain first the limitation of movement of the root of the tail (and so restricted movements of the free distal part); and, in subsequent stages, the position of the coccyx and the disappearance of the tail distally to it. An animal, one of the forbears of the orthograde type, finding it necessary to maintain its tail apposed in this way against the perineum, would soon find the tail of little or no use, or even a disadvantage; and the disappearance of the tail with the habitual or common assumption of the vertical position of the body is easily understood.

Such must especially have been the case when a vertical position began to be assumed by suspending the body from tree-branches by means of the extended fore-limbs and hands. This posture is freely assumed by Gibbons of this day (Keith[54]): and there is no doubt that by altering the pressure condition in the abdomen and pelvis (besides in other—but not in more important—ways), it played a great part in the evolution of the orthograde type. We indeed believe—as Keith has taught us—that when the power for its easy maintenance was established, the body structure had been so altered in adaptation to it, that it rendered possible the erect posture by standing when descent to the

earth occurred; and that when terrestrial life became common, the ultimate free and easy pose of man on his feet was assured. But if the increase of pressure in the pelvis, induced by such suspension, caused the tail to be so flexed that any free part of it was rendered useless; how much more must the increase of pressure and the impactions of pressure, produced by activity with the pelvis dependent, have necessitated such flexion! For just as in the Gibbon the posture of suspension is correlated with the remarkably rapid method of progression of that animal—caused by swinging or leaping from tree-branch to branch, using the fore-limbs and hands only for this purpose, by which great distances are covered (Keith)—so we may believe the pre-Gibbon moved. It is indeed more likely than not that this strange posture was itself determined (in the first case) by this manner of moving and was secondary to it—we can easily imagine how this movement was first accidentally performed during some time of stress by tree-dwelling forms (e.g. for escape). During such movement, the pressure in the pelvis must have been greatly increased: we have indeed but to think of the centrifugal force developed by such swinging movements to be sure of this.

In the position and the limitation of the movement of the coccyx, then, we have evidence that the phylogenetic regression of the pelvic floor occurred in this way. But the reason of this, at first, is not clear. At first we may think such change in the pressure conditions in the pelvis would have introduced little difficulty; and imagine that with the increased work but an hypertrophy of the muscles concerned would have occurred. But had this been so, the pelvic floor muscles in orthograde forms would be disposed in a way similar to those in pronograde mammals: that is to say, had their hypertrophy simply occurred, these muscles in man would still arise from the

pelvic brim and be but greater in mass than in pronograde forms. But we see that instead of becoming hypertrophied, these muscles regressed. The presence of increased work being allowed (for which as we have seen there is sufficient reason), the only explanation of this can be that the conditions of hypertrophy did not obtain. And to the factors at play, we must attribute the change of regression: we must believe it was because hypertrophy could not occur, and because the visceral pressure in the pelvis at some time during evolution became too great for the muscles that these muscles regressed.

That increase of work, when the conditions of hypertrophy do not obtain, causes regression of muscle becomes clear from a consideration of the changes which occur in the flank muscles of the abdominal wall during pregnancy. The weakening of these and the stretching of the linea alba, the result of the increased tonic contraction, especially of the transversales, arising reflexly in pregnancy, is sufficient evidence of this.* But I have no doubt the same explanation will make clear the regression occurring elsewhere †: it does so certainly in that similarly pathological regression of the pelvic floor which occurs as prolapse advances, and is to be seen in late stages of the disease.

Figure 3 gives an admirable picture of such regression. In it we see portrayed a state in which regression is in progress, not one in which an equilibrium has been re-established after regression has occurred. One sees a thoroughly fatigued pelvic floor. Not only is the muscle affected, but also its connective tissue attachments; its fascial continuations reaching to the ilio-pectineal line have stretched, and the white line anteriorly has sunk considerably. The condi-

* These changes are fully discussed in a paper by the author [75].

† E.g. in the psoas. ' The formation of tendon from muscle may be traced in the psoas magnus, for in the infant it is almost devoid of tendon " (Bland-Sutton [9]).

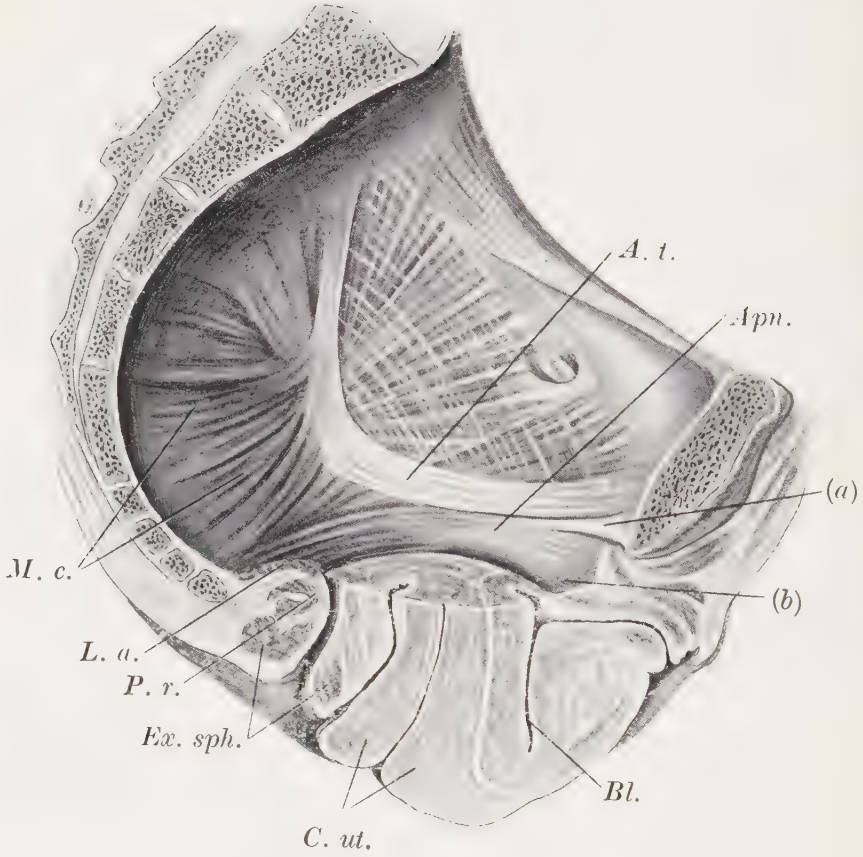

FIG. 25.—A SECTION OF A PELVIS IN MEDIAN SAGITTAL PLANE FROM A CASE OF PARTIAL PROLAPSE, SHOWING REGRESSION OF THE PELVIC FLOOR ANTERIORLY.

(From Halban and Tandler.)

M. c. = Coccygeus muscle.
L. a. = Cut surface of the post-anal part of the levator ani muscle.
P. r = Cut surface of a muscle, probably a part of the levator ani (? pubo-rectalis) modified by the condition.
Ex. sph. = External sphincter of anal canal.
C. ut. = Cervix uteri.
Bl. = Bladder.
A. t. = Arcus tendineus.

Apn. = Antero-lateral aponeurosis (or tendon) in which the levator ani ends. Part of this is attached to pubic arch (*a*); the other and larger part (*b*) passes inwards below the pubic arch, in front of the prolapsed mass, and across the median plane to become continuous with the corresponding part of the other side.

(*For Notes see opposite page.*)

[*To face page* 351

tion is exactly comparable with that occurring in the flank muscles in pregnancy. The musculature clearly has had too much work to do: it has been unable to hypertrophy, for the conditions on which hypertrophy depend, have not obtained. The condition has resulted from the endeavour on the part of the musculature to deal with the pressures in the pelvis, which though not greater than normal, or in the later stages not even so great, have been too much for the muscle. For as the condition advances, the muscle gets weaker and becomes more and more unable to cope with the pressures: only if the prolapse can be prevented from advancing (e.g. by reducing the pressure) can the muscle either maintain the state it now is in, or in more favourable cases be restored to its former condition.

In the figure facing this page, we have a different and earlier state of affairs (Fig. 25). In spite of the partial prolapse, the pelvic floor (with the obturator in place) is obviously structurally sound; and the position of the parts behind the aperture is as good as (or even better than) normal. A state of equilibrium seems to have been established and maintained. Evidence of this is seen in the

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EXPLANATORY NOTES (Fig. 25)

Note the well-marked arcus tendineus, occupying a normal position. The coccygeus is splendidly muscular. And the remains of the levator ani are so well developed that muscle fibres pass from side to side across the median plane behind the rectum, in a fairly thick and compact layer (L. a). Traced forwards, the posterior muscle fibres of this layer pass to the white line; the anterior ones end in tendinous fibres (Apn), some of which reach the pubic arch (a), others end in ligamentous tissue below it (b). These tendinous fibres represent part of the muscle which evidently has undergone regression. Those which pass from side to side below the pubic arch are a new formation: their presence is further evidence that during life the prolapsed mass was suspended by constriction.

Halban and Tandler, in their drawing (from which the above was taken) refer to the post-anal muscle layer as the pubo-rectalis. But it seems to me to be the remains of the ilio-coccygei which have taken on the function of the pubo-rectalis. The small piece of muscle seen in section below this layer and above the external sphincter of the anus (p. r.) may possibly be the remains of an altered pubo-rectalis.

As was to be expected, with such a pelvic floor the coccyx occupies its normal position. The post-anal part of the floor is even raised to a higher level than normal. The slight displacement downwards anteriorly of the floor is explained first by the initial injury sustained during childbirth affecting this part of the floor most; and next by the considerable descent of the bladder present in this case.

markedly muscular layer forming the post-anal part of the floor; and in the condition of the coccygeus. The arcus tendineus is well developed, and occupies its normal position. Thus, neither the white line itself, nor the fascia above it, has yielded; so that regression here has not occurred. But in the part of the floor guarding the aperture, the condition is different. Whilst the part behind, and postero-laterally, is well developed muscularly; antero-laterally, the muscular tissue gives way to tendinous or aponeurotic fibres, also well developed. It is obvious this part of the muscle has regressed. The reason of this is that in the earlier stage of the prolapse the work put upon the muscular boundary of the aperture, in the endeavour to restrain the prolapse, was too great. But subsequently —because of adaptive changes—the muscle became able to function efficiently; and by grasping and suspending the prolapse to prevent further extrusion. And, in this way—by bearing the brunt of the altered pressure conditions—the post-anal part of the floor has been preserved from being stretched and depressed, so that regression of this part has not occurred.

That in the early stages of prolapse a greater work than normal falls on the muscle still guarding the pelvic floor aperture, is obvious. For the muscle is at a disadvantage. The visceral extrusion acts as an expanding bag upon the margins of the aperture and causes the musculature forming the boundaries at the sides and behind to be exposed to pressures due to activity. Clearly, to withstand these, the muscle must respond to a greater extent—its tonic and other contractions must be greater—than when the aperture is not dilated in this way. To prevent further extrusion, the muscle must adapt itself to the new pressure conditions. But this takes time; and during this time (if the muscle is overworked) regression occurs. In the case before us, the regressive process has been stayed by the

response of the muscle—i.e. by the ability of the muscle to contract sufficiently. The muscular part has hypertrophied; and the aponeurotic part has similarly developed. Regression, here, has come to a standstill, just as during phylogenetic evolution (as viewed in normal man) it has also done so; but the relics of the regressive process in the former, as in the latter, remain.

But in many of the figures of the pelvic floor from cases of partial prolapse, given by Halban and Tandler,* such a condition as that displayed in Fig. 25 is not seen. In the majority, the white line is broader than normal and displaced downwards anteriorly to some extent—the condition being intermediate between the normal and that depicted in Fig. 3. In such, it is plain, regression of a much greater extent—simulating a different type—is in process. The supporting part of the pelvic floor as well as that constricting the neck of the prolapse are affected. In these cases, it is clear, the pelvic floor about the aperture has not been able to respond sufficiently to stay the extrusion: it has not been able to grasp the neck of the prolapse sufficiently, in spite of its persistent contraction, to maintain the intra-pelvic visceral parts in place. Not only are the parts within the embrace of the muscle able to expand the aperture to a greater extent than in the other case—because the contraction of the muscle is insufficient—and so to stretch the muscle and enlarge the aperture; but in consequence of this, more and more of the parts above come down, still further dilating the aperture and aggravating the process. Thus, more and more of the pelvic floor gradually becomes affected in this way; until, as the prolapse increases, the whole of the musculature becomes profoundly degenerated. It is because the muscle reacts to the pressure of the extruding parts and tends to resist

* The author knows of no such store as that found in Halban and Tandler's book

it that it suffers. Clearly, the blood supply to the muscle (that is, through the muscle), and the state of visceral metabolism, have a say in the reaction of the muscle: that is why in some cases an equilibrium obtains, why in others it does not occur, and devolution (regression) proceeds.

Our explanation of the regression of the pelvic floor muscles, then, is different from the general opinion. For us, the degenerative changes which occurred, which are peculiar to orthogrades and quite different from a simple atrophy —being indeed complicated by the persistence, even the increase of their function—were not caused by loss of the tail; but were caused by the reaction incurred in dealing with the visceral pressures in the pelvis, which had become greater. This reaction was so considerable—so intense—that it not only caused the root of the tail to become permanently flexed (giving the *raison d'être* of the position of the coccyx and an explanation—if one be wanted—why the tail disappeared); but also brought about degenerative changes in the pelvic floor muscles themselves which led to their regression. In other words, the degenerative changes were due to fatigue—a fatigue arising in these muscles from over-use, and which must have persisted, at least during active states, in a whole succession of individuals.

4. The Sufficiency of the Pelvic Floor

That this regression did not continue for always—that it came to a standstill—was due to other changes which subsequently occurred. In man, we have noticed two conditions at least, not present in pronograde mammals, which assist in the retention of the viscera—i.e. in the maintenance of the pressure conditions—in the pelvis: the one

is the lumbar lordosis; the other, a change in the pelvic floor itself—the appearance of the pubo-rectalis. The former directs sudden rises of pressure anteriorly against the anterior abdominal wall (facilitating the effect of sudden contractions of the diaphragm), and so preserves the pelvis from the full effect of direct impactions; the other more efficiently occludes the pelvic floor aperture so that impacts which reach the floor can be more certainly withstood. The pelvic floor, previously disadvantageously placed in respect of such impactions, as it is in the new-born babe, becomes by the recession of the pelvis which the lumbar lordosis caused, in a less disadvantageous position. And by the modification of its structure, whereby the pelvic floor aperture, from being a longitudinal slit extending throughout the length of the floor, becomes greatly shortened so that it is confined to the anterior part of the floor, a much more efficient means for meeting impactions exists. These adaptations, however, are only of importance during activity; for it is the rises of pressure which then are produced, which in man tend to cause the escape of viscera from the abdomino-pelvic cavity as in prolapse and hernia; and which in the pre-Gibbon must have been especially difficult to meet. During resting or static states, the opposing of the visceral pressure presents less difficulty: in man, it is easy enough; in the pre-Gibbon, it could hardly have sufficed of itself to have caused such regression of the pelvic floor as occurred.

But these adaptations are not peculiar to man. They are present to some extent in the Gibbon of to-day; and it is clear they began in the pre-Gibbon form, when progression by fore-limb activity (with the body suspended) was becoming habitual. Thus, we see that the lumbar lordosis in man is not due to the effect of gravity, as at first we might think; it is not the result of the weight of the body above on the lumbar spine: it is caused by

muscular contraction—by the tonic contraction of the erector spinæ, which was increased. This occurred (for one thing) because of the body suspension: the musculature (with others) had to sustain the weight of the lower part of the trunk and of the legs in the suspended position. But the lumbar lordosis also tended to preserve the pelvic floor from impactions of pressure, arising during activity, and which must have been specially intense when great activity occurred, e.g. locomotion with the pelvis dependent. And this seems to have been the more important cause; and is indeed a much more satisfactory explanation.

Similarly, the increased reaction of the pelvic floor muscles to the increased pressure in the pelvis caused the pubo-rectalis to evolve. In the Gibbon, this change is already advanced. An ano-coccygeal raphe is present in this animal; and according to Keith, nodules of cartilage are to be found in it, which represent the remains of the less proximal coccygeal vertebræ. This is evidence that as the tail was pulled forwards, the posterior attachment of the pubo-coccygeus, or part of it, slid forwards upon the tail-root towards the pubes.

It was the lower (the inferiorly placed) part of this, preserved by the upper part from the visceral pressures, which eventually became the pubo-rectalis. This displacement forwards of the inner margins and adjacent parts of the pubo-coccygei (inserted into the ano-coccygeal raphe (see Fig. 8, p. 32), and the appearance of the pubo-rectalis situated immediately below these muscles, with its freedom posteriorly from connections, is intelligible when we think of the increased contraction of the ancestral muscle in the evoluting form, and the preservation of its inferior part from pressure impulses from above. The relief of this lower part from strain, explains how this part did not regress—or, if it regressed, how its recovery was possible—so that it possesses no tendon

posteriorly, and arises in front from the pubic arch by muscle fibres. Instead of meeting the pressure from above and preventing the escape of pressure impulses by pressing against its fellow of the other side, as happens in carnivora when the muscles are taut (see Fig. 7, p. 29), it specialised by becoming, with the corresponding part of the other pubo-coccygeus, a U-shaped muscle, adherent anteriorly, by which in man the pelvic floor aperture is much more efficiently guarded.

But these were not the only changes which occurred. With the assumption of the erect posture—even of that caused by suspension—the transversales of the abdominal wall increased in mass. Keith has shown that in a Gibbon of the same weight as a pronograde monkey, the transversales are heavier than in the latter animal. That is to say, that the transversales hypertrophied with the evolution of the orthograde type. Similarly, the diaphragm altered: its crura developed, and it became more muscular. Thus, a greater intra-abdominal pressure came to prevail, and so a greater visceral metabolism. The nutrition of all the body parts therefore improved; and the pelvic floor muscles were not only supplied with more blood per unit of time, but with blood under a greater pressure than before, by which means the blood could be forced through the capillaries permeating the muscles more easily in spite of the increased tonic contraction of these muscles. Moreover, the muscles were supplied with a richer blood—with blood containing more oxygen, etc.; and with a purer blood—with one less laden with waste products. Such changed conditions clearly would affect these muscles; we know that in man the nutrition and developmental state of muscle depends entirely on such conditions.

It was the occurrence of such changes as these which stayed the regression of the pelvic floor and enabled the musculature to meet the visceral pressures in the pelvis

and the excursions which reached it, at first with less fatigue and subsequently easily. Equilibrium being thus re-established, the possibility for further advances such as we have indicated was laid open ; so that in man, in spite of its previous regression, the pelvic floor musculature suffices for his needs. But even though this explanation of the phylogenetic regression of the pelvic floor as given, and of its subsequent cessation, so that even an ascendancy of the musculature to a certain level of physiological reaction occurred, should be unacceptable ; yet must those who deny that this musculature in man functions efficiently, stand refuted at the persistence of its structure, and be confounded in the presence of its hypertrophy in pregnant and robust corpulent women. For what can be more certain, had the function of the pelvic floor muscles ceased with the loss of the tail, than that in present-day man these muscles would have disappeared quite ? Striated muscle—one of the highest of tissues—does not persist in the body inactive ; it rapidly disappears, when continuously unused. Nor does the neuro-muscular mechanism, with the habits of centuries, radically change its function, and come to move for other ends than formerly. The statement that this is so is an assumption, put forward to meet difficulties which authors have been unable to explain ; and is incongruous with the whole of biology. A tail-mover in a pronograde ancestor of man—even if we have to go back to amphibian times—is, if it persist, a tail-mover in man ; if in man that statement cannot stand, then the supposition that the muscle in his ancestor was tail-moving is shown at once to be erroneous.

The fact that striated muscle still persists in the pelvic floor shows that this musculature still possesses some function ; and that this is to oppose the visceral pressure is shown not only by all our previous argument, but also by the different conditions obtaining in the lower animals in

respect of the occlusion of their pelvic outlets and of their manners of living to which we have referred.

In normal man, the levator ani is a well-developed muscle: according to Piersol [79], the coccygeus itself (greatly neglected) is always conspicuously muscular. But the several segments of the pelvic floor, as we have noticed, are not equally well marked. It is not surprising that the part about the aperture, which has been especially developed to form with the pubic arch a sphincteric mechanism for the express purpose of guarding that aperture, should be easily palpable and that no doubt of its functioning—even by violent opponents—should exist. The part behind—the superficies—is much thinner; but this does not prevent it from functioning and from doing so efficiently. We have already given an explanation of this thinness in women with flat abdomen (p. 8); we have seen it is explained by physical laws. But in corpulent and in pregnant women, this with the other parts of the musculature are thicker, being remarkably well developed in robust people of these types. To such an extent does this occur that no one, after comparing its state (as by palpation) in such cases with its state in healthy women with flat abdomen, can doubt it to be hypertrophied.

It is for this reason that the musculature is best studied clinically in robust corpulent or in pregnant women. But of these two, the latter is to be preferred; because during pregnancy, the intra-pelvic fat gradually disappears, so that two fingers in the vagina, flexed laterally and depressed, come almost at once on the superior aspect of the musculature, the vaginal wall and some scanty connective tissue being only between: whereas in corpulent people, the fingers are separated by a layer of fat, more or less dense, as well. There is besides in the latter much more fat below the musculature, in the ischio-rectal fossa and adjacent

subcutaneous tissues. To palpate the musculature, the thumb has but to be placed outside, on the skin, in apposition to the tips of the internal digits. All the soft tissues comprising the pelvic floor, with the vaginal wall and some soft intra-pelvic connective tissue above, and the skin and subcutaneous tissues beneath, are now between the thumb and fingers.

If this examination be made in the region of the pelvic floor aperture in a well-developed primagravida,* say six or eight months pregnant, a tense mass of tissue, constricting the bases of the fingers against the pubic arch, will be easily perceived. This can be traced from its pubic origin backwards by the side of the vagina. On rectal examination, it can be identified; and (with fingers of the other hand simultaneously in the vagina) it can be perceived to pass, from beside the vagina, backwards by the side of the rectum and then inwards behind the gut, around it posteriorly, to become continuous with a similar mass of tissue on the other side. This, in turn, can be followed forwards to the pubic bone of its side, to which—one can satisfy oneself—it is attached.

This is no other than the pubo-rectalis (see fig. 6, p. 24). Lying at a higher level than the external sphincter of the anus—easily to be felt—and being therefore above the other superficial muscles as well (which are impalpable), it is readily distinguished from all these, surpassing them in mass. That this mass of tissue, which (in the erect posture) is more or less horizontally disposed and the lateral parts of which (in that position) plainly pass from before backwards, is muscular, is shown by the manner by which the fingers are compressed against the

* Choose a primagravida: for in multigravidæ the pelvic floor has already been injured by child-birth, and since the abdominal wall is similarly less well developed, the same degree of hypertrophic changes in the pelvic floor does not occur

pubic arch ; and, more particularly, by getting the patient to cough. If this be done when the tissue in question is fixed between the thumb and fingers, as explained, it will be felt to harden, i.e. to contract, revealing at once its nature. By moving the fingers within the vagina and the thumb externally more posteriorly, a thick fleshy mass of similar tissue will be found—in mid and late pregnancy it is easily discernible—passing from the ischial spines, which can be clearly identified, towards the coccyx and ano-coccygeal raphe. It also can be fixed by the examining digits ; pinching it, is painful ; on coughing, it also contracts and plainly is muscular.

By careful examination of the pelvic floor in this way, in numerous women, one gets a clear idea of its muscular nature ; and of the force of its activity in different women. What one feels is the muscle ; what one experiences is the force of its contraction. One is insensible of its fascial lining on which so much importance has been laid ; nor is one conscious of any other structure. Whilst valuable in that it reveals not only differences in the different parts of the floor, but differences in the floor as a whole in adaptation to different bodily conditions, it is besides the only method available for the student of medicine ; for whilst such clinical material as we have mentioned is at hand everywhere, who has seen the pelvic floor displayed by dissection in the pregnant woman ? It is perhaps true to say that no one has studied it in this way in such a case. I have never had the opportunity of doing so ; * nor in my reading have I come across any publication or reference to

* Since this was written such an opportunity occurred—but was missed. The case was one of eclampsia in a primipara for which I performed Cæsarean Section : the fits, however, continued and death ensued twenty-six hours later. I was, however, so engrossed with the disease that the condition of the pelvic floor was forgotten, nor do I know that I could have obtained permission to remove the pelvis.

the subject. The corpses of pregnant women do not find their way to the dissecting room; and under most conditions of practice they are difficult to deal with in this respect. There is an opportunity here for some Pathologist connected with a Lying-in Hospital to render Medicine this service. A primagravida, dying from eclampsia or from concealed accidental hæmorrhage, before delivery or after Cæsarean Section, would afford the best material; as the pelvic floor, not having been injured, would be displayed to its best advantage and would give unprejudiced evidence.* But this is not wanted to prove the existence of muscular tissue in the pelvic floor; even in ordinary cases it can be displayed, as every anatomist can testify.

But it is useless to try to arrive at an idea of the pelvic floor musculature in the normal by dissection in corpses of old and decrepid people; nor in those of the well developed, can one appreciate the effect of the muscular contraction. How great is the force evolved by living muscle can only be tested by examining the living; and only in this way can one come at a correct opinion. And that an hypertrophy does occur in the conditions we have mentioned becomes plain. Eden and Lockyer admit "that abnormal increases in intra-abdominal pressure may lead to compensatory hypertrophy of these muscles" [26]; but these authors do not state that hypertrophy occurs. Few authors refer to the possibility. Dickinson, however, has definitely described its occurrence.[20] But no reference to the work of others, nor any statement will — quite rightly—convince a reader. Actual experience is necessary; which, however, is easily obtained.

It is, as we have seen, on the activity of this muscular substance comprising the pelvic floor that the develop-

* Descent of the pelvic floor, especially that due to post-mortem relaxation, must be reckoned with.

mental state of itself and of its other parts—its connective tissue covering and its fibrous connections to the skeleton —depends. The high metabolic power of the striated muscle fibres and the influence of these on the other parts, is sufficient evidence and is explanatory of this. Given an adequate supply of blood, sufficiently enriched and freed from waste products, and an intact nervous supply, these cells explain much. They explain how the pelvic floor supports the pelvic pole of the visceral mass, and so understruts the " retinaculum uteri," even during resting states ; how it reacts during times of stress ; how it far and away surpasses the effect of the visceral connective tissue condensations under all conditions. But they explain much more. They explain how it is that the fibrous connections of the pelvic floor remain in position in spite of the strain put upon them by its unceasing activity ; they explain the position of the " white line," of the non-yielding of the obturator fascia—the aponeurosis by which the pelvic floor laterally is attached to the pelvic brim. They also explain how, with increase of work—the resistance of a greater visceral downthrust—an adequate blood supply being allowed—not only the muscular parts, but the other—the fibrous—parts hypertrophy, as in pregnancy; and how during ontogeny, an equilibrium at a high level is produced. We have already referred to all this ; and it is not strange. What is so strange is that the muscle should be regarded as degenerate ; and of little or no importance in the statics of the pelvic viscera.

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